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A METHOD OF CLASSIFYING AUDIOGRAMS.*

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INTRODUCTION.

A method of classifying audiograms plotted from the data obtained by the use of Western Electric Company audiometers will be described. The classifications given to audiograms by this method include in themselves no implications whatever as to any causes possibly responsible for deviations from perfect hearing. This method has been developed not as a matter of clerical convenience, but as a basic part of the major research program of this laboratory, which is the study in a strictly objective manner of all of the possible interrelationships between the facts obtained with respect to the hearing function, the several items of the past and the present medical history, the histopathology of the organ of hearing, age, race, sex, etc.

Demonstrations of the method to others who are using audiometers have convinced me that interest in such a method of classification is sufficiently widespread to justify publication at this time. The use of it in this laboratory for the past two years has demonstrated its practicability.

BASIS OF CLASSIFICATION.

Audiograms, as recorded on the commonly used chart form devised at the Bell Telephone Laboratories by Fletcher and associates, are linear graphs. The essential characteristics of any plotted line are

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form, direction and position with respect to its origins. These are the only elements which are taken into consideration in this method of classification.

Absolutely straight line audiograms are of very infrequent occurrence. Therefore, minor deviations have been disregarded, and the line has been considered with respect to its more general form, etc.

When thus viewed, almost all audiograms fall into one of the following groups as to general form or shape: *1.* those which are approximately straight lines; *2.* those which are approximately straight lines except for a marked local projection downward or upward; *3.* those which consist of two, or sometimes three, major segments, each part being relatively straight but having definitely different directions; *4.* those which are markedly concave upwards throughout much of the middle part, this curved portion becoming continuous with straight segments at one or both ends. The general direction on the chart of the approximately straight line audiograms (Groups 1 and 2 of above) may be *a.* horizontal, *b.* ascending gradually from left to right, *c.* descending gradually from left to right. The line forming the left part of those audiograms with two or three major segments (Group 3 as to shape) may run in any one of the three general directions just listed for straight line audiograms; the right hand segment (or the middle segment in those cases with three parts), however, always descends from left to right and with a sharp inclination. In those audiograms of this group which have three segments, the third, or right hand, one is either horizontal or, sometimes, slightly upwards to the right.

The above described elements are considered as primary in this method of classification, and the major classes thus determined are designated by the first six capital letters of the English alphabet, or by combinations of these (see below). The major classes are subdivided on the basis of the position, or level, on the chart of the whole or of the component parts of the audiogram. Thus degrees of loss of acuity or extents of regions of differential loss are regarded as secondary in this system of classification. These features are indicated by arabic numerals used as subscripts to the capital letters. Within each major class, the larger the subscript number used, the greater is the amount of hearing loss indicated.

DESCRIPTIONS OF CLASSES AND SUBCLASSES OF AUDIOGRAMS, AND DEFINITIONS OF THE LIMITS OF EACH.

These descriptions and definitions are primarily for the data which may be obtained with the W. E. Co. 1-A audiometer. The frequencies of the twenty tones that can be generated by this instru-

ment, and the maximum intensity available at each pitch, are shown in Fig. 1. The definitions are also applicable, with only minor modifications, to audiograms made by the use of the W. E. Co. 2-A audiometer. Certain features of these modifications will be indicated later.

Those audiograms consisting of approximately straight lines running horizontally across the chart are divided into two major classes, termed A and B, respectively. In general terms, Class A represents good hearing for all tones, and Class B more or less uniform loss of acuity for all parts of the tonal range heard.

Class A: Requirements for inclusion in this group are: *1.* all tones must be heard; *2.* the general position of the line of the audiogram must be above the line on the chart which represents twenty sensation units loss.

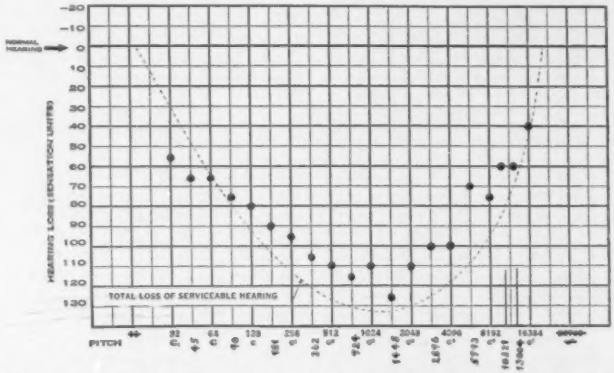

Fig. 1. The frequencies of the twenty tones produced by the Western Electric Co. 1-A audiometer and the maximum settings possible for each tone.

Three subclasses, A_1, A_2 and A_3, are made of this group, indicating respectively that the line of the audiogram is: *1.* near the zero line of the chart; *2.* near the ten sensation units loss line of the chart, and *3.* nearer the line of twenty sensation units loss than that of ten. In the subclass A_3 are included cases in which the hearing acuity for one or more individual tones falls to twenty-five, provided that the general impression given by the audiogram is that of a line averaging better than twenty (see Fig. 2).

Many individuals who do not hear the tone of c^7 (16,384∞) when produced at the maximum intensity of the audiometer with the receiver against the ear in the usual manner, can nevertheless hear the tone distinctly at even a decreased intensity of production

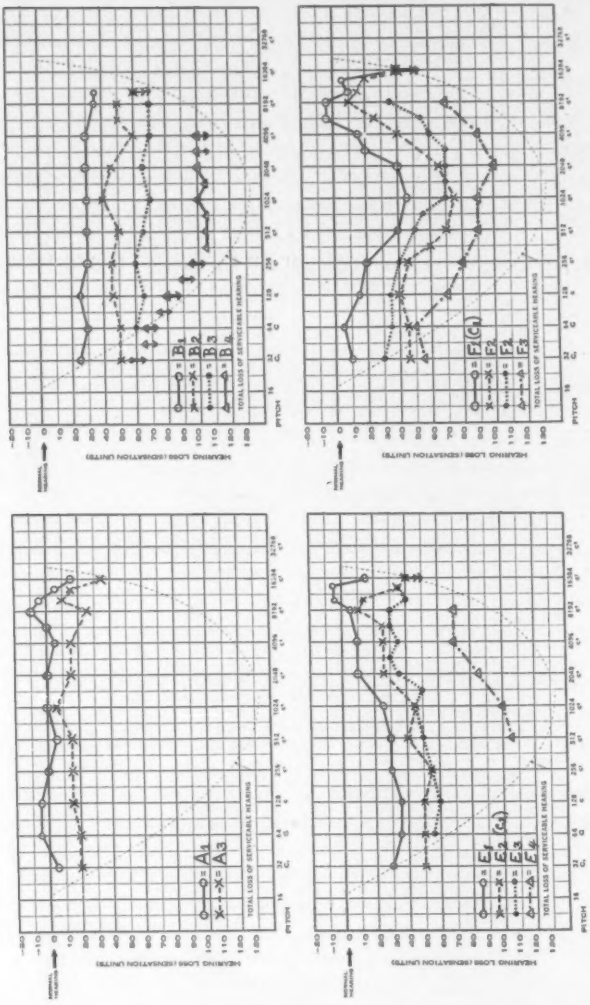

Fig. 2. Actual examples of audiograms which have been classified as indicated.

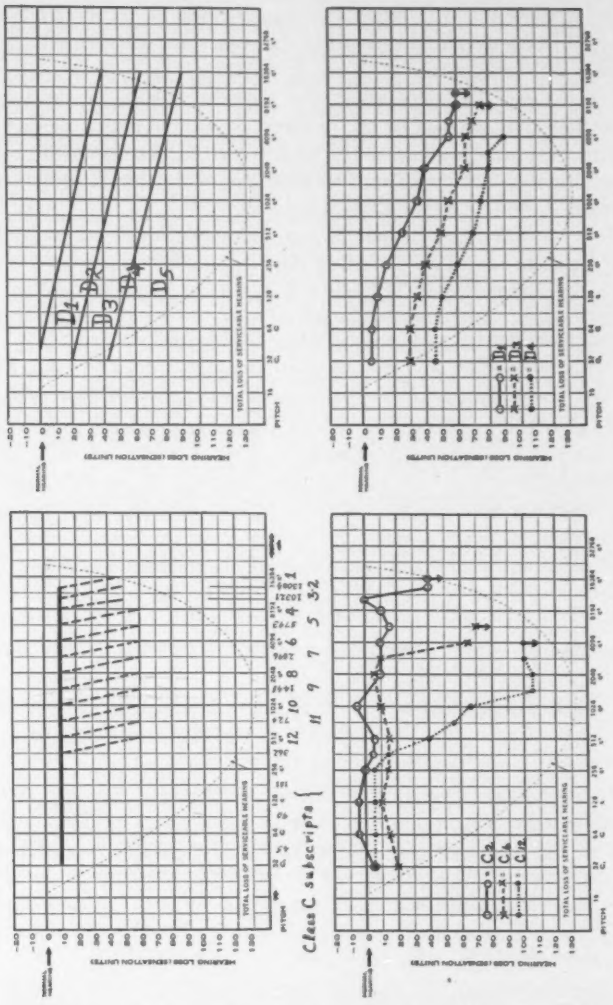

Fig. 3. Left: Diagram showing the twelve possible subclasses of Class C audiograms, and actual examples of three which have been classified as indicated.

Right: Copy of the ruled chart used in determining the subclassifications to be given audiograms of Class D, and actual examples of three which have been classified as indicated.

when the distance of the receiver from the ear is increased slightly. This indicates that the effect of "standing waves" is an important factor in the acuity of hearing for this tone. Its wave length in air is such that individual variations in the dimensions of the external auditory canal doubtless affect the apparent acuity of perception when the ear is covered by the receiver. For these reasons, provided that the frequency of 16,384∞ was perceived, its threshold value shown on the chart is given but little weight in deciding upon the subclass to which an audiogram belongs.

Class A with Dip: An audiogram with a marked local projection downward, but otherwise meeting the requirements above stated for Class A, is regarded as belonging in this class; and is classified as an "A with a dip," the frequency of the tone at the apex of the projection being indicated. Thus: "A_2 with 4096 dip" (see Fig. 4), "A_3 with 8192 dip," etc. Rarely there are cases with two "dips."

Class B: Approximately horizontal lines across the chart at some level below the line indicating a loss of twenty sensation units characterize the audiograms of Class B. Such records reveal a more or less uniform loss of acuity of tonal hearing. In order to group together those with roughly similar degrees of such loss, the area on the chart below the zone for Class A is regarded as divided into four horizontal bands, with their common boundaries just below the lines on the chart which indicate losses of 40, 60 and 80 sensation units. The zones are numbered from above downward, and audiograms falling within one of these zones are termed B_1, B_2, B_3 or B_4, respectively (see Fig. 2).

Total deafness, irrespective of cause, is included in this general group and termed B_5. The total absence of hearing is regarded as representing a uniform loss for all tones.

The deviation of acuity for individual tones by as much as ten sensation units beyond the boundary of the given zone in which most of the audiogram falls is not taken into account in the classification given. A local projection of more than ten units, however, either upwards or downwards, is recorded as a "peak" or as a "dip," respectively, in the same manner as dips in Class A. Thus: "B_1 with 4096 dip," "B_2 with 1024 peak," etc. (see Fig. 4).

When the intensity is progressively increased the so-called threshold of feeling is reached sooner for both the higher and the lower tones than for those of the middle range. This is a well established fact. Marked individual variations in the intensity required to reach this threshold have been recorded, but the degree of loss of hearing acuity probably plays no part in determining the intensity beyond

which an apparent perception may actually be a sensation of feeling. An approximation of the average intensities required to excite this threshold of feeling for the frequencies in the range of hearing is represented on the commonly used audiogram charts by the line labeled, "Total Loss of Serviceable Hearing." It is based on the work of Wegel[1] and others.

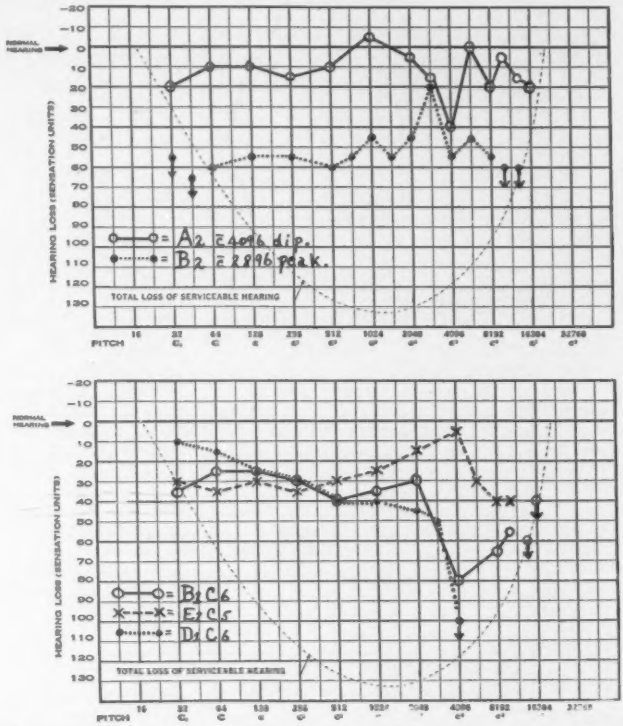

Fig. 4. Upper: Actual examples of audiograms with a dip and with a peak, and the classification given.
Lower: Actual examples of three different combinations of classes.

It is, therefore, evident that even if the lowest and the highest of the tones could be produced at an intensity setting of, for instance, 75 decibels greater than the intensity represented by the "zero" of the chart, the apparent "hearing" which might be manifested would not necessarily be indicative of the auditory acuity

1. Wegel, R. L.: The Physical Examination of Hearing and Binaural Aids for the Deaf. Proc. Nat. Acad. Sc., Vol. 8, No. 7, July, 1922.

of the ear being examined. It is essential that due allowance be made for this factor in classifying the audiograms of Class B, and of some of the other groups. Limitation of the "field of hearing" (*i. e.*, a lowered upper limit and a raised lower limit) is usually obviously present in cases with B_2 and B_3 audiograms, and is so marked in those falling in the B_4 zone that at most only two or three

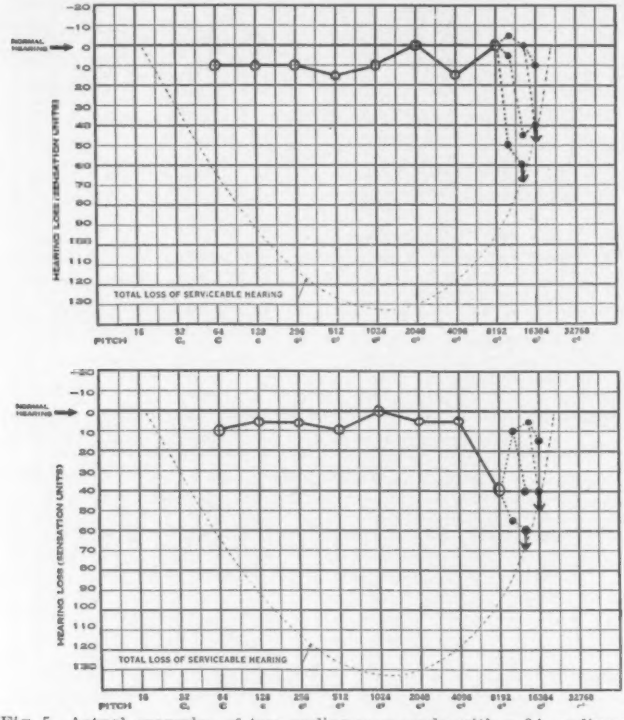

Fig. 5. Actual examples of two audiograms made with a 2A audiometer, with diagrammatic extensions showing the possibilities that the use of a 1-A audiometer might have revealed. See text matter.

octaves of the middle of the tonal range can be acoustically perceived with certainty.

There are, of course, some audiograms which are on the border between A_3 (or C_1, see below) and B_1. In these cases consideration is given to the possible disturbing effects of conditions at the time of the examination, particularly the presence or absence of noises and the general health of the patient.

Class C: The line of each audiogram of this group has either two or three major segments; the left hand one is horizontal and in the zone of Class A audiograms, the next segment descends abruptly towards the right (at least twenty and usually more than thirty sensation units in an octave),[1] and in the cases with a portion of the line continuing to the right of this descent this part may be either horizontal at the level of the end of the descending element or it may ascend somewhat, but does not again reach the level of the left hand component. Such audiograms represent good hearing for all tones up to some given frequency with a rapid drop in acuity for

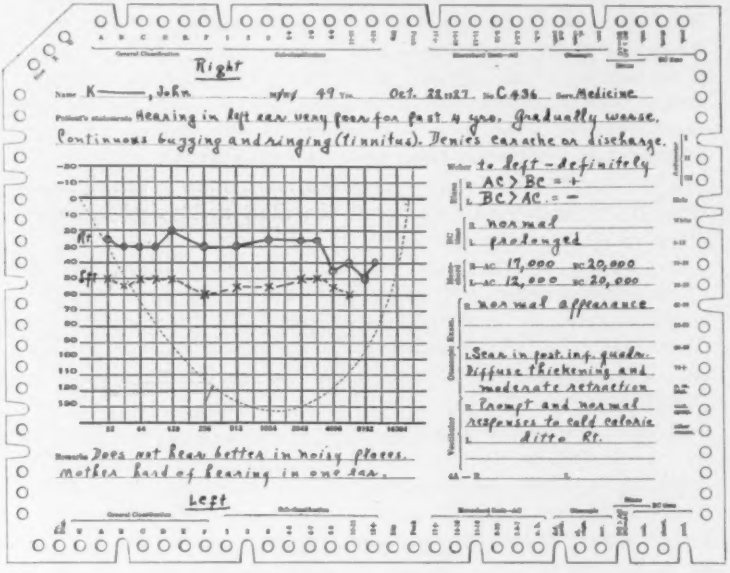
Right
Name K———, John
Oct. 22nd 27
C436
Medicine
Hearing in left ear very poor for past 4 yrs. Gradually worse.
Continuous buzzing and ringing (tinnitus). Denies earache or discharge.
to left - definitely
AC > BC = +
BC > AC = —
normal
prolonged
AC 17,000 BC 20,000
AC 12,000 BC 20,000
normal appearance
Scar in post. inf. quadr.
Diffuse thickening and moderate retraction
Prompt and normal responses to cold caloric
ditto Rt.
Does not hear better in noisy places.
Mother hard of hearing in one ear.
Left

Fig. 6. Facsimile at reduced size of the border punch card used for indexing purposes (front side).

the tones just above this frequency and with all tones above this region of abrupt loss either poorly heard or not perceived at all at the maximum intensities with which each can be produced by the audiometer. More of the audiograms made at this laboratory belong in Class C than in any other one.[2]

1. Sometimes the difference in acuity of hearing for tones only a half-octave apart is as much 70 sensation units.
2. Cf. first three pages of article by Guild, Crowe, Bunch and Polvogt; Correlations of Differences in the Density of Innervation of the Organ of Corti, etc. Acta Otolaryngologica, Vol. 15, 1931. Also Ciocco, elsewhere in this issue of "The Laryngoscope."

The subscript used in the divisions made of this class indicates how many of the tones produced by the audiometer are poorly heard or not perceived. On this basis it is necessary to make twelve subclasses of Class C in order to include all of the audiograms of this general type which are in the laboratory's collection. A diagrammatic representation of the twelve subclasses is given in Fig. 3, left upper. Illustrative of the definitions for each of these subclasses is that for a C_5 audiogram:—the frequency of 5793∞ poorly heard, all higher tones poorly or not heard, but 4096∞ and all lower tones well heard. Substitution in this definition of the frequencies which can be selected from the diagram yields those for each of the other subclasses. In Fig. 3, left lower, are shown actual examples of a C_2, a C_6 and a C_{12} audiogram.

Class C with Dip: An audiogram with a marked local projection downwards in the Zone A segment, but otherwise meeting the requirements for Class C, is classified as "C with a dip," the same principles of designation being used as with Class A.

Class D: Approximately straight line audiograms which descend gradually from left to right across the chart are typical of those classified as D, with the exception of the subclass D_0 (see below). In audiograms of this general description the rate of descent shows considerable variation, both in respect to the average per octave and in the differences between adjacent tones. This fact makes the determination of rational limitations for the subclasses more difficult than with the other major classes, and the natural consequence is that there is more often an uncertainty as to which subclass a given audiogram belongs in than with the other classes. The method which has proven most satisfactory in practice for subclassifying this group is that of superimposing the audiogram to be classified over an arbitrarily ruled chart (see Fig. 3, right upper) and determining which line or zone it fits best for the major part of its course: D_1, D_2, D_3, D_4 or D_5, respectively.

There are cases in which the threshold values for all tones up to 1024 or 2048∞ are in the range given for Class A, while beyond this region the line of the graphic record descends gradually to the right on the chart, with a rate of descent resembling that characteristic of the typical Class D audiograms rather than the abrupt type of drop which characterizes Class C. These have been tentatively included in Class D and have been designated by the subscript "zero."

There are also audiograms at every level throughout the range of Class D in which the line shows the tendency to have a convexity upwards in the region of the middle range tones, although the general course of the whole graph is that of a gradual descent. It is

possible that these, together with the D_0 cases, really belong in a separate class and differ fundamentally from the typical Class D audiograms; for the present they have all been grouped together. The difficulties encountered in assigning many of the "D" audiograms to a definite subclass tend to indicate that this class is not as "homogeneous" as the others.

Class E: Approximately straight line audiograms which ascend gradually from left to right across the chart are typical of those classified as E. The lower pitched tones are not heard as well as the higher ones. The rate of ascent varies considerably in different audiograms, as does also the location of the high point of the curve. The right hand (high range tones) end of the audiogram may be horizontal or it may descend rapidly: if horizontal, the audiogram is nevertheless classified as an E; but if it descends rapidly, as is usually the case, the audiogram is given a combined classification E,C—which will be explained later.

The audiograms of Class E and the "E" elements of the combined ones are subdivided into four subclasses on the basis of the following definitions (see Fig. 2).

E_1—low tones heard at intensities indicating from 20 to 35 sensation units loss, with higher tones in the range of Class A audiograms.

E_2—low tones around 40 to 60 on the chart, with higher tones in the range of Class A. The two lowest tones—32 and 45 cycles—may not have been heard at all.

E_3—low tones similar to those of E_2, with high tones distinctly better heard than lower ones, but not running up into range of Class A at any point. (The rate of ascent of an E_3 audiogram is less than that of an E_2 audiogram.)

E_4—the lowest tone which was heard with certainty — usually 90 cycles, because of the threshold of feeling factor discussed above —around 65 to 80 on the chart, with the successive higher tones distinctly better heard.

Class F: Audiograms belonging to this class are characterized by a marked depression in the middle part of the graph. This shape of the curve is due to the fact that for several of the middle range tones the number of sensation units of loss is greater than for those tones both higher and lower. The extent of the region of depressed acuity is distinctly greater than in the cases of the marked local projections downwards termed "dips"; and also "dips" are usually at frequencies of higher pitch than the lowest part of the concavity of the F audiograms.

Three subdivisions of the class are made on the basis of the following definitions (see Fig. 2).

F_1—middle range tones perceived at intensities around 30 to 40, with both lower and higher tones at a threshold in the range of Class A audiograms.

F_2—middle range tones perceived at intensities around 50 to 70, with both lower and higher tones heard better by at least 20 sensation units.

F_3—middle range tones at 75 or lower, with both lower and higher tones at least 20 sensation units better.

Because of the different ratios between the intensities required for the "threshold of 'normal' hearing" and for the "threshold of feeling" in the several parts of the tonal range, and the design of the chart form used, the Class F audiograms come more nearly representing a uniform percentage loss for the intensity range of hearing for the several tones than do those of Class B. In the latter the loss of acuity is "uniform" in the sense of the number of sensation units loss for each tone. When the data of a Class B audiogram are expressed in terms of percentage of total range for each tone, the loss of acuity is relatively least for the middle of the tonal range. Further elaboration of this topic is out of place in this article; it has been mentioned merely to emphasize the necessity of constantly keeping in mind the basic facts back of the commonly used diagrammatic representations of data regarding the hearing function; failure to do so can easily lead one far astray in the interpretation of the actual data.

COMBINATION CLASSES.

There are audiograms in which certain of the dominant characteristics of each of two of the major classes are present. Such audiograms are regarded in this method of classification as combinations and are designated by the respective two letters and the appropriate subscripts of each. The nature of the left hand component of the audiogram is indicated by the first letter and its subscript, and that of the right hand component by the second letter and its subscript.

The right hand component of almost all of the combination audiograms descends abrupty, being thus similar to the characteristic feature of Class C. That is, up to some given frequency the graph has the essential characteristic of one of the subclasses of a B, D, E or F audiogram, while just above this frequency there is a rapid descent of the line on the chart towards the right. The subscript used for the C element is determined in exactly the same way as for the simple Class C audiograms, and thus indicates how many of the higher tones produced by the audiometer are more poorly heard than would have been the case had the entire audiogram been of the type indicated by the nature of the left hand component. It is un-

necessary to list all of the combinations which have been found. With the principles of classification above described in mind, it should be easy to visualize the appearance of other possible combinations from the following illustrated examples: B_1C_6, D_1C_6, E_1C_5 (see Fig. 4). The Class C audiograms are of course on this basis in reality "A, C" in nature; and it is convenient at times to regard them thus, even to the point of specifying the subclass of A to which that element belongs.

"UNCLASSIFIABLE" AND UNRELIABLE RECORDS.

The audiograms in this laboratory which do not belong to some one of the classes which have been described form a very small percentage of the collection. These have been designated for the present as "Unclassifiable" (meaning, of course, in this system) and are designated by the letter U. When for any reason, such as lack of intelligence or co-operation on the part of the patient, the record is very unreliable, it is either classified as an "X" or the apparent classification of the audiogram is supplemented by the addition of the letter X.

USE OF THIS METHOD OF CLASSIFICATION WITH THE 2A AUDIOMETER.

The Western Electric Co. 2-A audiometer provides for the production of the octave interval tones from 64 to 8192 cycles. This is an octave less than the 1-A audiometer at both ends of the tonal range, and the intervals between tones are twice as large.

Class C is the only one in which the subscript numbers would be changed by the direct application of the definitions for the 1-A audiometer to the 2-A instrument. If the number of high tones of the 2-A audiometer which are poorly or not heard is indicated by the subscript, it is for each region of the chart less than the number of yet higher tones produced by the 1-A audiometer. Both of these audiometers are used in this laboratory, and in order to avoid confusion in the discussion of the records the plan has been adopted for Class C and for the C element in combination classes of using the subscript number which corresponds to the tones produced by the 1-A audiometer rather than the smaller number produced by the 2-A. The audiometer which has been used is indicated for all classifications given the audiograms by the use of Roman numerals as a prefix: I for the 1-A and II for the 2-A.

It is obvious that examinations with the 1-A audiometer reveal defects in hearing at either end of the tonal range of an extent too small to come into the field covered by the 2-A audiometer. Therefore, in the consideration of audiograms made from data obtained with the latter instrument, the following possibilities must always be kept in mind.

a. Any II A audiograms might have been, had the larger audiometer been used, either a I A, a I C_1, a I C_2, or a I C_3 (see Fig. 5, upper). Only in those cases with a very low upper tone limit, as determined by other means, is it possible to be reasonably certain to which one of these four possibilities the hearing acuity represented by a II A audiogram really belongs. Therefore, the percentage of audiograms classified as A will always be larger in collections of audiograms made with the smaller audiometer than when the larger one has been used.

b. Likewise, examination with a 2-A audiometer groups together as II E_1, the types I E_1, I E_1C_1, I E_1C_2 and I E_1C_3.

c. Likewise, I F_1, I F_1C_1, I F_1C_2 and I F_1C_3 become grouped together as II F_1.

d. The classification II C_4 signifies that the highest tone (8192∞) produced by the 2-A audiometer was poorly heard, and that all the tones lower than this were perceived in the range of Class A. Since no data can be obtained with this instrument regarding the acuity for the three highest tones produced by the 1-A audiometer, the possibility exists in all such cases, except when the upper limit is shown by other means (monochord, Galton whistle, etc.) to be much lowered, that the defective hearing for 8192∞ is due to a "dip." It does not necessarily signify that the acuity of hearing is also poor for the yet higher tones. Therefore, the audiograms made with the larger audiometer and classified as "I A with 8192 dip," "I C_1 with 8192 dip," "I C_2 with 8192 dip," and I C_4 all get grouped together as II C_4 when the 2-A audiometer is used (see Fig. 5, lower).

e. Since the acuity for the half-octave intervals cannot be determined with the 2-A audiometer, the possibility is always present that any decline in acuity did not begin until at least a half-octave higher than the last tone which was well heard. For example, a II C_6 might be either a I C_6 or a I C_7, etc.

f. For the same reason, "dips" and peaks" which occur at or near the half-octave frequencies cannot be discovered by the use of the 2-A audiometer. Therefore it is reasonable to expect that in any large series of examinations with this audiometer the number of audiograms showing these marked local projections will be less than in a similar series made with the 1-A audiometer. (Naturally the use of continuous tone range audiometers should reveal a yet larger percentage of records with dips and peaks, and permit a more exact determination of the beginning of the "decline" in the Class C records.)

ADDENDUM.

Inclusion of Other Features of Examination and of Medical History : 1. A Struycken monochord is used routinely in this laboratory for the determination of the upper limit of hearing by both air and bone conduction. The value of this for air conduction, expressed in thousands of cycles per second, is appended to the classification given the audiogram, thus grouping more closely together those cases with similar hearing. For instance, the upper limit in cases with a C_8 type of audiogram may be anything between almost normal and "monochord not heard"; this latter meaning that the upper limit is less than 5500∞.

The side—right or left ear, or bilaterally similar—is added to the notation made on the record by using the letters, R, L, or RL. Examples of the full form of the notation, which includes all of the items thus far mentioned (and which is being routinely used in this laboratory), are: I A_2-18-R, II C_6-10-RL, I C_2 with 4096 dip-16-L, I E_1C_4-14-RL, II C_8-n.h.-L, I D_2-unk-R, etc. (n.h. = monochord not heard; unk. = monochord not used). The reading of such notations for a given case, after one has become familiar with the method, immediately brings to mind the appearance of the audiogram (the upper limit with the monochord helping in this), and whether or not there is any marked difference between the two ears.

2. Any fairly complete otological examination of course includes many other items of importance. When one attempts to add symbols for all of them to the audiogram classification, the resulting notation is too unwieldy for convenient use. Also, the number of possible combinations becomes so large and the possible ways of filing each record so many that it is necessary to resort to an elaborate system of cross-indexing or to a so-called punch card method in order to bring together from a large collection those cases with similar records for any given number of features. The method which has been adopted in this laboratory for assembling together all of the facts from each case history, in a form convenient for statistical studies, is that of a so-called "border punch card." By this means those cases with any desired combination of conditions can be quickly selected for study. The form used, as filled out for a particular case, is shown in reduced size in Fig. 6.* On the side shown is

*The cards are 8½ x 11 inches, and are of high quality thin card stock. Because of the large amount of handling which they receive, and the danger of tearing out the holes in the process of sorting, "toughness" was especially considered in selecting the stock for the printer to use. The trade designation of that used is Resistall Index white, 110 lb. The "blank" cards have all of the round holes cut by the printer; it is especially important that this be so accurately done that all of the corresponding holes in each card are placed so that the sorting rod will pass through without obstruction due to poor alignment of the holes. A special punch is used to cut out the marginal side of each hole that is indicated by the data of the particular case.

placed an abstract of the otological data, including a copy of the audiogram, and on the other an abstract of the rest of the medical history. Provision is made along the upper and lower borders (respectively for right and left ears) for indexing the classification given the audiogram, the air conduction upper limit for the monochord, the results of the three classic fork tests, and the condition of the tympanic membrane. Along the right border of the same side are the spaces used to record the particular audiometer used, race, sex, age (in decades), the patient's statement as to past history of otitis media, and the presence or absence of vestibular symptoms. The spaces of the remaining border are labeled from the opposite side. Each of these is used to record the presence or absence in the medical history, either past or present, of any of the diseases that belong to the designated group. The number of spaces available limits the number of groups of diseases which can be separately indexed. The diseases and groupings to which indexing positions have been assigned are: scarlet fever; typhoid, malaria, diphtheria; cardiovascular; brain tumors; other neurological; syphilis; carcinomas and sarcomas; nephritis; anemia, leukemia, etc.; arthritis; endocrine organ diseases; tuberculosis; gastrointestinal; lower respiratory tract (non-tbc.); poisons; focal infections of the nose, throat and mouth; other focal infections.

SUMMARY.

1. A method of classification for audiograms plotted from the data obtained by the use of the Western Electric Co. 1-A audiometer is described.

2. Certain general principles of audiometry and facts concerning hearing acuity are emphasized in describing the basis for the classification groups made.

3. The adaptation of the method for use with the Western Electric Co. 2A audiometer is described; also how proper comparisons of these records may be made with those from the larger instrument.

4. The purpose of the method described is to facilitate the objective study of these records of hearing acuity from the standpoint of possible correlations with all or any of the other facts known about the cases, past and present medical history, aural and systemic pathology, age, race, sex, etc.

5. An index card of the "border punch" type, on which these classifications and numerous other items of interest can be recorded in convenient form for statistical studies, is described.

OBSERVATIONS ON THE HEARING OF 1,980 INDIVIDUALS; A BIOMETRIC STUDY.*

ANTONIO CIOCCO, Baltimore.

Introduction: The object of this paper is to present certain data derived from a statistical analysis of the hearing of 1,980 individuals tested in our laboratory. These individuals were all patients in the Johns Hopkins Hospital and were hospitalized for ailments other than otological. Therefore, on the basis of their clinical diagnoses and the past medical history we have especially sought whether or not there was any systemic condition or disease that influenced their hearing. Theoretically this problem is simply thus: Given the measure of hearing and certain systemic conditions, is there association or interdependence between any of these conditions and impairment of hearing?

We wish to note that we do not speak of finding the cause of impairment of hearing. To find the etiology or cause intended in the strict significance given by Mills (*i. e.*, antecedent), cannot be undertaken with statistical analysis alone. What is intended by statistical analysis? It is the application of the Method; that is, pure logic applied to the analysis of a mass of observations utilizing certain theorems of the mathematical theory of probabilities. It has for its ultimate object that of attaining a knowledge of the order in the manifestation of phenomena by determining their distribution, movement and interrelations. In other words, it serves as a substitute for direct experiment when this is impossible, or to extend the experimental results so as to give them universality, by giving the means to calculate the limits within which any final conclusion may properly be made. And finally, it constantly emphasizes the avoidance and elimination of the Baconian Idols from conclusions; those errors of bias, prejudice and ignorance.[1]

We have thought it necessary to outline the above principles for the benefit, not of those who abhor, distrust and eschew statistics, but for those who believe in any result printed in numerals and bearing the heading: Statistics. Too often have we seen these principles disregarded in medical works and in such a way as to render valueless otherwise excellently conducted observations.

*From the Otological Research Laboratory, Johns Hopkins University.

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MATERIAL AND METHOD.

In order to secure material for study that is unselected with respect to hearing, one phase of the routine work of the laboratory is the examination of the hearing of as many of the patients in the hospital as the facilities of the laboratory make possible. This is done by co-operation with the staffs of the several medical and surgical services of the hospital, who designate which of the patients in their care are in suitable condition to be so examined. The selection being in this way not at all influenced by previous knowledge of the hearing, one can affirm that this material truly represents a random sampling. The 1,980 cases analyzed in this study represent only part of the material collected and specifically the hearing examinations made from September, 1928, to August, 1931, with the Western Electric Co. 1-A audiometer. Those tests done with the other audiometers used in this laboratory have not been included so as to avoid the possible introduction of variations due solely to difference in individual instruments.

The otological examinations consisted of the audiometric test; Weber, Rinné and Schwabach tests with a 512 d.v. fork; determination of the upper limit of hearing for both air and bone conduction by means of a Struycken monochord; and otoscopic observations. Examination of the vestibular function has had to be limited usually to caloric stimulation only.

In spite of the prominence given to the audiogram in this laboratory, it is well realized here that the audiometric examination is not a substitute for the well established fork tests, but does give additional information of value in diagnosis. S. J. Crowe has well expressed this, "We cannot rely on audiometer tests alone for diagnosis."[2]

To handle the material conveniently and quickly a punch-card system of indexing was introduced and utilized. The card used was that described by Guild in the preceding paper in this journal. On one side it has all the information regarding the hearing, each ear being shown separately, and regarding the audiometer used, sex, age, race, otitis media history and vestibular symptoms, if any. On the opposite side of the card is annotated the clinical diagnosis and past medical history of the patient.

The audiograms were classified according to the method described by Guild. This was done not for clerical purposes alone, but as the first step in this research. The reasons were two: First, it must be realized that in routine examinations one cannot hope for precise threshold values. Besides errors due to condition of audiometer and

external conditions that are rendered as constant as possible, important factors in determining the values of thresholds are the intelligence and co-operation of the patient, and his physical condition. The examiners seek by repeated checking to reach the "true" threshold but in reality it can only be considered an "average" value and experience shows that it may vary insignificantly within the limits of ± 5 to $\pm$ 10 db. It is clear then that by the creation of "zones" we avoid those errors that might be simply due to conditions of the individual at time of testing and receive also a clearer impression of the entity of the raising of threshold. The second reason is that the audiogram in this way is considered as the "profile" or physiognomy of the hearing ability of the patient and with one symbol this is fully described, avoiding the enormous labor of giving the threshold value at each frequency.

An important benefit that is derived from the above method is that one is not tempted to average the two ears in order to represent the hearing of an individual. The use of the average or "composite" curve would not produce an error of any magnitude if the hearing in the two ears were practically the same. But if they are not, the average is perfectly meaningless and completely false. We have used in this study as the measure of hearing for each individual the value of the thresholds of his "worse" ear, considering this the best expression of the individual's auditory condition. A simple example will render this clear. Given an individual with a unilateral conductive lesion, and with an audiogram showing for the affected ear thresholds of 40 db. for all frequencies and for the nonaffected ear hearing thresholds at the "zero" line, the average or "composite" curve is at 20 db., which is considered fairly good for our conditions. Taking then a group of individuals with the same lesion and impairment, one would have to conclude that the audiograms show that the lesion in question does not affect hearing, which is obviously untrue.

As it has been said, the examiners carefully and repeatedly check the patients to arrive at an accurate value of threshold. Sometimes, either the mental or physical conditions are such that the response is entirely unreliable; in this material there were 107 cases of unreliable tests and these were eliminated, as also 26 cases in which the audiograms were unclassifiable with this method. This latter group constitutes only 1.4 per cent of the material and shows the practicability of the method. Our total is thus reduced to 1,847 cases. Tabulating this data according to age and sex and types of audiograms, as in Table I, we observe: First, that the C class is the most common and that with advancing age the "modal" hearing pro-

gresses from the A to the C_6 and C_8 types, which appear to be the extreme "normal" limit; second, that there is a striking difference between males and females when one considers the incidence of C_6, C_8 and D_1 types; third, that the 4096 "dip" is found the most often and appears to be associated more frequently with C_2 and C_3 curves, and it is obvious that the incidence of this combination increases with advancing age.

THE PROBABLE SIGNIFICANCE OF THE 4096 "DIP."

Leaving aside for the present any comment on the age and sex differences, we wish to observe closely the 4096 dip. Drury stated that in cases with a dip at 4096 the existence of a luetic taint might be regarded as probable.[3] Of this we have found no evidence, but from our observations we think it is the first sign of a degenerative change in hearing which may continue until the hearing curve has reached the C_6 or C_8 stage, which is the most common for advanced age. This conclusion that we cannot yet prove to our complete satisfaction is based upon the following facts:

First, four cases in which tests at later dates show the changes that are here illustrated (see Fig. 1). The first three cases have similar alterations in hearing, consisting in the appearance of a dip at 4096 d.v. that was not present before. (The improvement of low and middle tones that is present in Cases 2 and 3 is certainly attributable to the better condition of testing in the second instance.) Case 4 had a 4096 "dip" when first tested, his second test showed a drop to a C_6 type. (It is well realized that four cases are not sufficient to prove that the loss of acuity for high tones takes place in the above manner, but this laboratory has been in existence only a relatively brief period and these represent all the cases of the few repeated tests to show significant change in acuity for high tones.)*

Second, excepting cases of traumas, certain intracranial conditions and conductive lesions, we have found that all individuals have similar general types of hearing curves on both sides. But, in cases of high tone loss, when they are not entirely similar, we find, for example, cases with C_6 or C_8 on one side, and C_1 C_2 and C_3 with 4096 dip on the other. This we interpret to mean that the latter individuals were tested in a moment when one ear was in an advanced state of degeneration with respect to the other.

Third, the fact that with respect to the males we find a relatively inferior number of females with C_6 and C_8 types of audiograms and, also, a significantly smaller number of female individuals with

*Two more cases similar to the first three have been found among the re-examinations made since this manuscript was submitted.

TABLE I—DISTRIBUTION OF TYP

Age	Sex	A—typical	A c 4096 dip	A c other dips	C_1—typical	C_1 c 4096 dip	C_1 c other dips	C_2—typical	C_2 c 4096 dip	C_2 c other dips	C_3—typical	C_3 c 4096 dip	C_3 c other dips	C_4—typical	C_4 c 4096 dip	C_6 and C_7	C_8 and C_9	C_{10} and C_{11}	C_{12}	D_0	D_1	D_2	D_3
0-19	M	24	3	3	8	1	4	14	2	1	3	1		3		1				1			
	F	29		3	9			5						1		1				1	2		
20-29	M	25	6	2	36	4	5	28	9	4	6	4		7		5	1			5	3		
	F	40		5	30			21	2	1	4	1				3				1	1		
30-39	M	12	3	2	17	7	1	23	25	2	9	8		3		13	7		1	5	5	5	
	F	12	1	1	37	2	1	38	5	1	13		1	6		5	2		1	4	3		
40-49	M	3	1		7	5	4	34	19	8	12	17	3	9	4	37	24	2	1	8	9	2	
	F				13		3	38	5	5	14	4	2	6		9	1			3	8	1	
50-59	M				1			11	13	1	4	17	1	11	1	36	30	6	5	6	8	6	
	F	1			2	1		21	2	4	7	5	3	17	4	10	7			8	7		
60-69	M								2	1	2	7		4	1	30	26	5	2	1	11	10	
	F						1		2	1	4	1		5	1	9	6			2	4	3	

N OF TYPES OF AUDIOGRAMS BY AGE AND SEX.

D_1	D_2	D_3	D_4 and D_5	B_1	B_2	B_3	B_4 and B_5	B_1C_4 and B_1C_5	B_1C_6 and B_1C_7	B_1C_8 and B_1C_9	B_1C_{10} and B_1C_{11}	B_2C_4 and B_2C_5	B_2C_6 and B_2C_7	B_2C_8 and B_2C_9	B_2C_8	E_1	E_2	E_3	E_1C_1	E_1C_2	E_1C_3
2				2 4	2 2		2 1	1 1								5			1 3		
3 1		1 1	1	13 6	5 3		2 2		1	1			1			3 4	2 1	2	1 3	2 3	1
5 3	5	2 1	3 1	15 8	5 5	1	3 5	2	6 2	1		1	1 2			2 4	1 1		3 2	6 3	1 2
9 8	2 1	5	2	22 11	6	2 2	9 3	1 2	4 1	4	2					1			1	3 7	3 2
8 7	6	1 3	7 1	18 10	6 2	2 1	4 3	7 2	11 1	5 1	3				1		1 1			2 5	2 1
11 4	10 3	4 1	5 3	3 7	4 4	2 2	2 2	3 2	6 2	5 1				3			1				

E_1C_3	E_1C_4 and E_1C_5	E_1C_6 and E_1C_7	E_1C_8 and E_1C_9	F_1	F_2	Total
					1	83
				1	1	64
	1	2		1		186
1			1		1	138
1		1	1		2	203
2		1	1			173
3	2	3		1		274
2	1				1	148
2	2	4	1			234
1			1	2	2	135
		3	1			144
		1	1			65

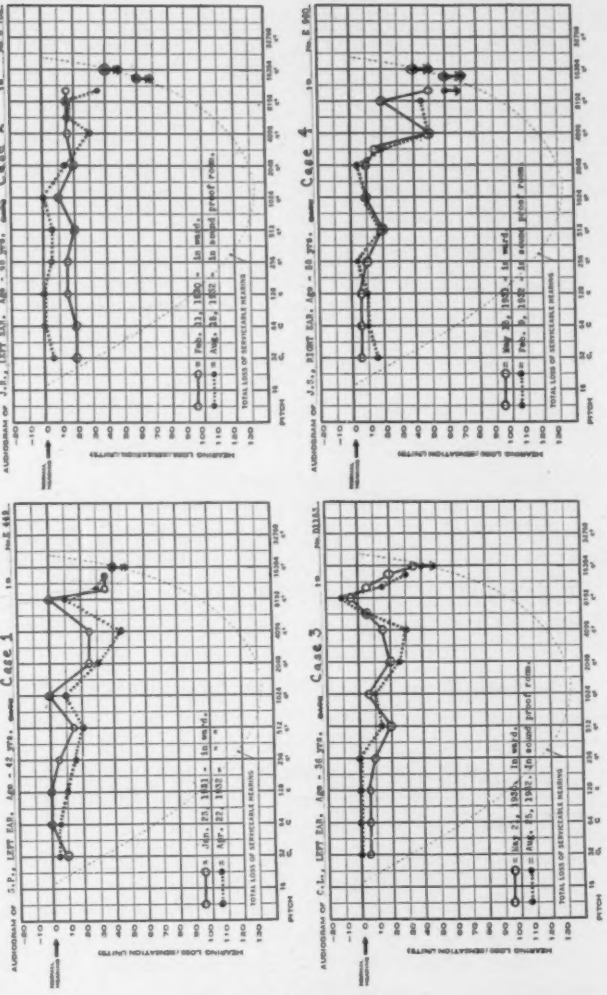

Fig. 1. See text matter.

a 4096 dip. This is seen in Table II, in which the comparison has been made between the number of males and the number of females having 4096 dips relative to the total number of cases, and more significantly, to the total possible cases, *i. e.*, cases in which the 4096 dip is possible, these being the A, C_1, C_2, C_3 and C_4 types of audiograms.

TABLE II—PERCENTAGES OF 4096 "DIPS" TO TOTAL CASES.

Age	0-19	20-29	30-39	40-49	50-59	60-69
No. of Cases—Male	83	186	203	274	234	144
Female	64	138	173	148	135	65
Percentages —Male	8.4	12.4	21.2	16.8	13.2	7
Female	0	2.2	4.6	6.4	8.9	6.2

PERCENTAGES OF 4096 "DIPS" TO TOTAL POSSIBLE CASES (A TO C_4 TYPES).

Age	0-19	20-29	30-39	40-49	50-59	60-69
No. of Cases—Male	67	136	112	126	60	17
Female	47	104	117	90	67	15
Percentages —Male	10.5	16.9	38.4	36.5	50.8	59
Female	0	2.9	6.7	10	17.9	26.6

Fourth, a study now nearing completion by Polvogt and Guild[4] on the histopathology in cases with high tone loss indicates that the cochlear area which they believe to be associated with the 4000 frequency may show degeneration together with, or independently of, the rest of the lower half of the basal turn.

THE BASIS OF ASSEMBLING THE TYPES OF AUDIOGRAMS INTO NINE GENERAL GROUPS.

In proceeding to analyze the data in Table I, it was immediately observed that the classification used divided our apparently large material into so many types that when age and sex were taken into account the numbers were so small as to make any attempt at a comparison of the different elements impossible. To remedy this in part we decided upon a more general grouping of types. Besides, there also arose the question of how to evaluate the intensity of raising of threshold. For example, a C_6 curve may have the thresholds for 4096 at 40, 60 or 80 db., and these differences may mean something and should be taken into account. Especially so since Polvogt[5] has found that in the majority of cases the pathology is severe in the region believed to be associated with 4096 d.v. when the audiogram shows a threshold at 60 db. or more for this tone.

On the basis of these facts and other histopathological observations made in the laboratory, and taking care also not to confuse

those cases with a negative Rinné test (that is, evidence of a marked conductive lesion) from those in which the Rinné was positive, we have classified our material into nine general groups that are here described.

I. Cases with good hearing or very slight high tone loss. This includes the A, C_1, C_2 and C_3 types of hearing, and the C_4 when the threshold for 8192 does not exceed 30 db. (see Fig. 2).

II. Slight loss for high tones. In this group are the C_4 type with 8192 above 30 db., and all other C and D types in which the threshold for the 2048 and, or, 4096 frequencies is above 25 db. and below 45. In this group are also included all cases with dips at the above frequencies and within the same limits for intensity (see Fig. 2).

III. Moderate loss for high tones. Like II except that threshold values are above 40 and below 65 db. (see Fig. 2).

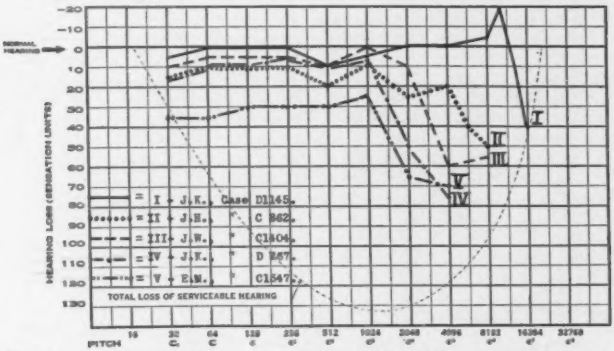

Fig. 2. Actual examples of audiograms falling in groups indicated.

IV. Severe loss for high tones. Like II and III except that threshold values are above 60 db. (see Fig. 2).

V. Slight loss for low and middle range and severe for high. The Rinné found positive. These include D_2, D_3, B_1C_4, B_1C_6, B_1C_8, B_1C_{10} and B_1C_{12} types (see Fig. 2).

VI. Extreme loss of hearing. Comprising the B_2, B_4, B_5, D_4 and all the combinations of B_2, B_3 with C's. The Rinné in these cases is practically impossible to judge, because the loss of hearing is so marked that the forks are heard only for a few seconds, if at all.

VII. Negative Rinné cases excluding those falling in Group VI.

VIII. Loss for low and middle range with high tones only slightly affected and with a positive Rinné test. This includes the B_1 and

B_2 types, and E_1C_1, E_1C_2, E_1C_3 (for the E_1C_4 and higher, we follow II, III and IV).

IX. The same as VIII with the exception that the *Rinné is unknown.*

We wish to emphasize that this grouping is a relatively rough gradation of audiograms based in part upon the pathology found in cases with similar types of hearing curves. We shall use it until our knowledge has progressed sufficiently to permit us to more accurately relate the pathology to the decrement of hearing as measured by the audiometer.

Our material tabulated according to the above groups and respectively for age and sex gives the following distribution (see Table III).

TABLE III—ABSOLUTE DISTRIBUTION OF HEARING GROUPS FOR AGE AND SEX.

Age		I	II	III	IV	V	VI	VII	VIII	IX	Tot.
0-19	Males	51	12	4			2	2	6	6	83
	Females	44	6				1	3	6	4	64
20-29	Males	100	35	10	7	2	3	9	11	9	186
	Females	99	10	2	1	2	3	6	12	3	138
30-39	Males	66	39	31	13	6	7	14	18	9	203
	Females	103	25	5	1	6	7	5	10	11	173
40-49	Males	66	66	53	27	8	12	11	20	11	274
	Females	69	36	7	1	3	6	3	15	8	148
50-59	Males	20	38	61	37	17	14	10	16	21	234
	Females	38	38	21	3	4	5	3	13	10	135
60-69	Males	2	18	45	33	21	13	5	3	4	144
	Females	3	20	14	2	6	7	4	6	3	65
Total		661	343	253	125	75	80	75	136	99	1847

But, as our interest is primarily to study the relation between systemic diseases and hearing, it is logical so as to achieve the results of having unselected population to exclude those cases in which the probable cause of hearing loss was definite or doubtful. This means excluding Group VI, composed of 20 cases of cerebellopontine angle tumors, 12 cases of impairment following traumas, 16 cases following acute fevers, and the rest having Meniere's syndrome or chronic otitis media or probable otosclerosis, etc. We exclude Group VII, all those in which the Rinné test is negative, since the conductive lesion is of such entity as to obscure other effects. And Group VIII because the slight loss for low and middle range was found probably due to extraneous conditions: receivers, audiometer, noisy wards. We could not use Group IX because of the fact that with the Rinné unknown the existence of conductive lesions could not be excluded.

Besides these cases there were others in the other groups in which the loss of hearing was attributed to traumas, occupation, or acute fevers, and these were excluded also. Therefore, the remaining cases are those in which whatever impairment of hearing there is, the etiology is unknown (see Table IV).

TABLE IV—ABSOLUTE DISTRIBUTION OF HEARING GROUPS FOR AGE AND SEX.

	Males						Females					
Age	I	II	III	IV	V	Tot.	I	II	III	IV	V	Tot.
0-19	51	11	3			65	44	6				50
20-29	100	35	10	6	2	153	99	10	2		2	113
30-39	66	34	29	9	3	141	103	22	5		3	133
40-49	66	63	47	15	2	193	69	35	7	1	1	113
50-59	20	37	55	33	11	156	38	37	20	3	3	101
60-69	2	17	42	30	16	107	3	20	14	2	4	43
Total	305	197	186	93	34	815	356	130	48	6	13	553

THE ACUITY OF HEARING IN RELATION TO AGE AND SEX.

U. Presbyacusia, or loss of high tones due to age, has long been known. In 1891, Zwaardemaker[6] observed the decrease with age in the perception of the upper limit as measured with the Galton whistle. Struycken,[7] in 1913, observed the same with his monochord and his figures compare very well with ours. Bunch,[8] in 1929, was the first to show the same with the audiometer. Our findings confirm his and, moreover, show that apparently the "normal" limit for loss of acuity is at 2048 or 4096. This is seen clearly in Table V showing the relative figures for each age-group. We ob-

TABLE V—PERCENTAGE DISTRIBUTION FOR AGE AND SEX, FROM ABSOLUTE FIGURES IN TABLE IV.

	Males—Hearing Group						Females—Hearing Group					
Age	I	II	III	IV	V	Tot.	I	II	III	IV	V	Tot.
0-19	78.4	17	4.6			100	88	12				100
20-29	65.3	23	6.5	3.9	1.3	100	87	8.7	1.7	.9	1.7	100
30-39	47	24	20.5	6.4	2.1	100	77.5	16.5	3.8		2.2	100
40-49	34.2	32.5	24.5	7.8	1	100	61	31	6.2	.9	.9	100
50-59	12.8	23.7	35.3	21.1	7.1	100	37.5	36.5	20	3	3	100
60-69	1.9	16	39.2	28	14.9	100	7	46.5	32.5	4.7	9.3	100

serve that the most common group (Mode) for the youngest age-group is for both males and females our Group I. The same is true of individuals between 20 and 39 years of age. For the males of 40-49 the mode lies in between I and II, but for the females it is still I. For those of 50 and above, Group III is the most frequent

for the males, while for the females it is not until the seventh decade is reached that even Group II becomes dominant.

For the monochord we find a lowering of the upper limit (air conduction) by about a thousand d.v. for each decade above the 20-29 age-group, and as Table VI shows, the mode for the last age-group

TABLE VI—AGE DISTRIBUTION OF PERCEPTION OF THE UPPER LIMIT OF MONOCHORD BY A.C. (EXPRESSED IN THOUSAND CYCLES).

Age	17+	14-16	11-13	8-10	5.5-7	Not Heard	Total	Mean
0-19	47	49					96	16.5
20-29	112	114	12	1			239	16.2
30-39	50	170	29	1	2	1	253	15.5
40-49	24	187	66	3	2	1	283	14.4
50-59	4	110	106	12	6	1	239	13.2
60-69		29	76	23	4	2	134	11.8
Total	237	659	289	40	14	5	1244	

falls in the 11-13,000 d.v. class. (The reason why we have not separated the sexes will be seen later.) Now we ask, is the loss of acuity for high tones gradual with increasing age, or is it due to an infection occurring some time in life, causing the impairment of hearing and after which the hearing remains stationary until the end? Although both hypotheses are permissible at present, we are more favorably inclined towards the latter because the great variation in the distribuition of groups for each decade appears to us more than what can be attributed to "normal" differences between individuals. Our impression is therefore that the increase in the percentage of individuals having moderate to severe loss of hearing with advancing age is none other than the expression of the increasing probability to contract whatever may be the cause the longer the span of life.

2. Bunch and Raiford[9] found that with advancing age the loss of acuity for frequencies above 2048, as measured by the audiometer, was more marked for the males than for the females. This we find true both as expressed by the incidence of 4096 dips and by the distribution for groups. To show in a more accurate way how marked this difference is we use the simple method of comparison that we shall here describe in detail because it has served us also for our other analyses.

The problem is placed in the following terms: Given the probability that a male of a certain age will have a certain type of hearing curve, and given the age distribution of the females, the resultant of combining the two gives a theoretical distribution of the females that should not be significantly different from the actual distribution, if in general there is no difference in hearing due to sex. If it is

desired to determine more accurately whether or not the differences between the two distributions are significant, one can apply Pearson's chi-square test and so reach a mathematical expression of the probability that they represent samples from the same population.[10] It should be said that we introduce by the above described method certain elements of error which must be taken into account in the interpretaion of our results. And these are principally three that we here enumerate. First, we have used for each decade the percentages as the value of the probability, and this would be correct only if the number of cases were very large. Second, the number of cases, for example, of individuals having a certain condition may be small and therefore our theoretical values may vary within wide limits. Third, the fact that, especially for the females, in our two last groups, IV and V, we have very few cases and so the measure of probability as found by the chi-square test will be exaggerated.

TABLE VII—ACTUAL FEMALE DISTRIBUTION COMPARED WITH "EXPECTED" ON BASIS OF THE MALE DISTRIBUTION.

	Hearing Group				
	I	II	III	IV	V
Expected	228.5	134.1	117.3	55.1	19
Actual	356	130	48	7	13

$X^2 = 156$ P = .000000 (Table for Biometricians and Statisticians by K. Pearson.)

In Table VII the total actual distribution of the females is compared with the theoretical values based upon the distribution of the males of the same ages. The probability of the two distributions being samples from the same population is theoretically less than one in a million. Very small, indeed, and proving the marked difference already apparent from a simple inspection of Tables I and V. For the upper limit of air conduction as determined by the monochord we do not find any such difference between the two sexes. The age distribution is almost identical. The males have a mean age of 41.3 with a standard deviation of 14.3 years, the females 39.4 and 14.1. The mean value, expressed in thousand cycles, is for the males: 14.55 ± 1.61; for the females: 14.77 ± 1.43. The probable error of the difference between the two means is equal to 0.088 and three times this is 0.264, which is greater than 0.22. Therefore, this difference is not significant.

Since Bunch and Raiford published their observations, which our study confirms, no one has yet apparently sought the reason why the males show a loss of hearing at 4096 and above more frequently

than the females. It may be a true sexual difference. But, on the other hand, although we have attempted to exclude all individuals whose occupational history was such as to give reason to suspect its influence on the hearing, still it must be remembered that the male patients who comprise our material are for the most part laborers whose several occupations may have exposed them in many instances to injurious noises; correspondingly, the females are, in general, housewives and have led a more sheltered life.

THE EFFECT OF MIDDLE EAR INFECTION ON HIGH TONE ACUITY.

It is well known that an otoscopic examination does not always reveal signs of past infections of the middle ear, so it remains for the examiner to elicit from the patient as much as possible concerning past otological ailments. But the statements of the patients are to be taken *cum grano salis;* from our records, we find that their memories go no further back than about fifteen years on the average. Nevertheless, we have attempted to see whether individuals who gave a history of healed otitis media together with those giving negative histories, but whose tympanic membranes showed marked pathology, were distributed significantly different as regards hearing groups than those with negative histories and normal or slightly changed tympanic membranes. From an inspection of Table VIII,

TABLE VIII—HISTORY OF OTITIS MEDIA AND DEFINITE PATHOLOGY OF TYMPANIC MEMBRANE.

	Males—Hearing Group						Females—Hearing Group					
Age	I	II	III	IV	V	Tot.	I	II	III	IV	V	Tot.
0-19	10	3				13	6	2				8
20-29	13	4				17	14	3				17
30-39	11	4	7	2		24	22	6	2			30
40-49	14	15	11	3		43	10	9	6			25
50-59	3	5	11	10	5	34	6	7	3	2		18
60-69		2	7	5	3	17	1	2	4	0	1	8
Total	51	33	36	20	8	148	59	29	15	2	1	106
Expected values on basis of cases without otitis media:												
	48.9	39.3	35.7	18.9	5.2	148	68.3	24.1	9.1	.6	3.9	106
	$X^2 = 2.68$		$P > .5, < .7$				$X^2 = 11.38$		$P > .02, < .05$			

comparing the total actual distribution of the former with the total theoretical values based on the distribution of the latter, there appears to be no significant difference. Using R. A. Fisher's abbreviated table,[11] the probability that they are not different is between 0.5 and 0.7 for the males. This means that, given a large population with a known distribution for hearing groups, were we to select at random a great number of individuals and distribute them

according to their hearing, and repeat this operation one hundred times, in at least fifty instances we would expect differences from the known distribution as great as those shown in Table VIII. For the females, the value of P (probability) is between 0.02 and 0.05, which is not significant according to the criterion of Fisher. Its low value is undoubtedly due principally to the third error, of which we

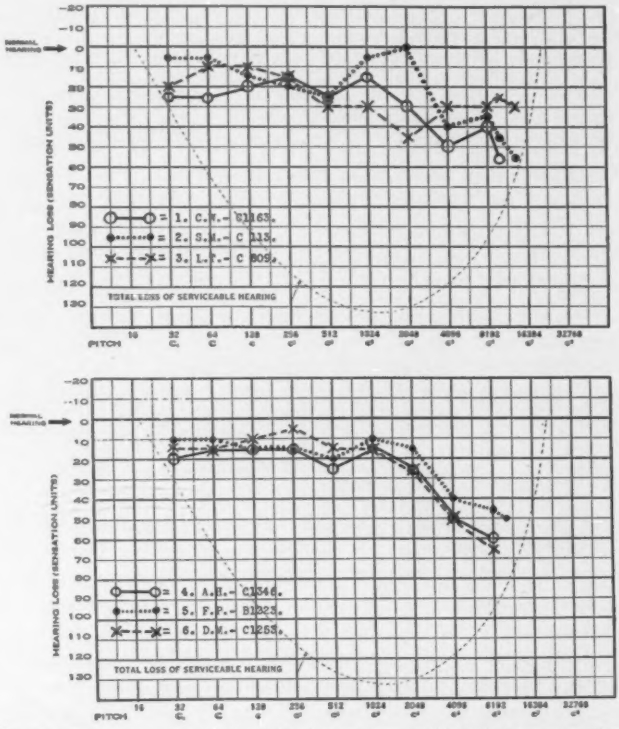

Fig. 3. Six cases of unilateral high tone loss with histories of unilateral otitis media.

have already spoken. From this evidence we should say that in general high tones are not affected by middle ear infections, were it not for other facts observed. First eleven cases of *unilateral* high tone loss with Rinné positive and with definite history of otitis media on the side with the high tone loss. Unfortunately, there were otoscopic examinations of only six of these; their audiograms are reproduced in Fig. 3. Otoscopically, the pathology of Cases 1. 2

and 3 was practically the same: discharge in the external auditory meatus and perforation of the tympanic membrane. Cases 4 and 5 had large calcification plaques on the tympanic membrane. Case 6 showed tympanic membrane thickened, with a granulation in the anterior lower quadrant and foul-smelling discharge. The opposite ears had good hearing and were negative as to any evidence of otitis media. The second fact that we have observed is that when we take all the individuals with a history of past middle ear infection together with those having chronic otitis media and compare their upper limits for air conduction, as determined by the monochord, with those of individuals having normal or apparently normal middle ears, the mean, expressed in thousand cycles, is for the former 13.75 ± 2.61, and for the latter 14.89 ± 1.69. The difference of 1.14 is greater than seven times the probable error of the difference, this being 0.159. Therefore, we must conclude that middle ear lesions do in some degree affect acuity for high tones. If this lowering of the upper limit were simply due to mechanical interference we should find a greater difference between air and bone conduction for those with middle ear lesions than for those without. Instead we find the same mean difference. And this can also be deduced from Struycken's observations[12] since he found no difference in hearing measured by the monochord before and after removal of cerumen plugs or incision of normal tympanic membrane. Nor did he find any significant difference before and after treatment of otitis media, catarrhal, nonperforative.

Therefore, if our data and method are at all to be relied upon we feel justified in believing that middle ear infections can produce degeneration in the inner ear. And this was apparently also the opinion of Politzer,[13] who, speaking of inner ear deafness, said: "It was known to early writers that disturbance of hearing (internal ear) occurred in the course of affections of the stomach, liver, kidney and uterus. *On the whole, according to my experience, this is much seldomer the case than is generally supposed, for I have often found in cases in which there is generally supposed to be a connection between the organic disease and the ear affection that the fundamental cause is palpable changes in the middle ear.*" Wanner[14] found that acute inflammations of the middle ear frequently affected the inner ear; and also that catarrhal forms affected the upper tone limit more than the suppurative forms. Struycken[15] cites an interesting case of chronic suppurative otitis in which after a year and a half from the time of treatment the monochord had descended from 15,000 to 7,500 cycles. It is for the pathologist to confirm or reject

this interpretation and the proof can be sought by measuring the degree of correlation between the presence of traces of previous inflammation of the middle ear and that of degeneration in the internal ear, keeping in mind that the membrana fenestrae rotundae is at least semipermeable and that its thickening and scarring can be considered a protective device of Nature to safeguard the internal ear.

THE INFLUENCE OF CERTAIN SYSTEMIC DISEASES.

The criterion used in selecting the diseases whose possible influence on hearing we have studied, was that of sufficient number. It resulted in our analyzing the association between disease and hearing only for those having a diagnosis or history of cardiovascular diseases, syphilis, carcinomas and sarcomas, tuberculosis (males only), gastrointestinal diseases (males only), or goitre (females only). The number of individuals giving histories of having had scarlet fever, malaria, typhoid or diphtheria was sufficiently large for our purpose, but it was felt that the results of an analysis would have little value because of having had to depend only upon the memory of the patient as to whether or not he had had the disease in question. For the diseases studied we rely upon the clinical diagnosis only, confirmed in a few cases by autopsy, though in all cases, as for syphilis especially, a careful objective survey of the history has been made.

It must not be forgotten that it is possible for an individual to have any combination of the diseases mentioned, and therefore only by a series of eliminations can one find which of these is the common element that in the majority of cases places the individuals in particular hearing groups. The method of comparison used has been described above, and in the following analyses the comparison is between those that are "positive" for a certain disease and those that are "negative." And we seek to find whether or not the distribution, by hearing groups, of individuals having certain diseases is different from that of individuals of the same age but not having the disease in question.

The principal preoccupation was, from the beginning, that of having enough material so that any conclusion reached could be considered definite, but the necessity of taking into account the age and sex factors has so fractioned our numbers as to raise a serious doubt regarding the significance of the results obtained. Before proceeding with our analyses it must be emphasized that any conclusion reached signifies that *in general* individuals having a certain disease more frequently fall into certain hearing groups, but with our means we

cannot exclude or deny the possibility that those diseases for which our findings show no association may in individual cases affect the hearing.

Cardiovascular Diseases: The group, our largest, is composed of 187 males and 111 females distributed in the following manner for age and hearing (see Table IX). In the young group we find more

TABLE IX—CARDIOVASCULAR DISEASES.

	Males—Hearing Group						Females—Hearing Group					
Age	I	II	III	IV	V	Tot.	I	II	III	IV	V	Tot.
0-19	8	2	1			11	7	2				9
20-29	5	3	1	1		10	15	1				16
30-39	11	4	6		1	22	13	2	1		1	17
40-49	12	11	7	5		35	12	11	3			26
50-59	5	17	17	9	6	54	3	12	8	1		24
60-69	1	8	18	20	8	55	3	6	7	2	1	19
Total	42	45	50	35	15	187	53	34	19	3	2	111
Expected values on basis of cases without C.V. diseases:												
	46.6	41.2	59.5	27.6	12.1	187	63	31.5	11.4	.9	4.2	111
	$X^2 = 5.02$		$P > .2, < .3$				$X^2 = 12.9$		$P > .01, < .02$			

commonly a diagnosis of chronic rheumatic endocarditis and aortic insufficiency; in the old group, arteriosclerosis and hypertension with myocardial insufficiency. Arteriosclerosis has from time to time been considered a cause of high tone loss. Perfectly plausible, but as yet not proven in a satisfactory manner pathologically, it may be so. But it is noticeable that most of the observers have studied only patients of advanced age, as usual forgetting to compare with individuals of like age without arteriosclerosis. Bunch[16] did not fall in this error and his results were negative as to association. Ours are also. Of course, there may be error due to the fact that arteriosclerosis may not have been considered by the clinician if the patient were in the hospital for other primary condition. Yet we cannot escape the fact that most young individuals with arteriosclerosis have good hearing.

Comparing the distribution of those having cardiovascular diseases with those of the same age groups who have not, we find that without question for the males there is no significant difference. But for the females one could not be so sure if it were not due to the second and third errors of which we have already spoken.

Syphilis: Luetic involvement of the auditory nerve is a well known manifestation. We asked whether in general those having syphilis had a more marked loss of acuity than those without. Our syphilitic patients had the hearing group distribution shown in

Table X; comparing their hearing with that of those showing no evidence of this disease, we find them essentially similar.

Taking C. N. S. syphilis alone, we reach the same conclusion (see Table XI).

Gastrointestinal Diseases: These consisted principally of ulcers of the stomach, duodenum, etc.; carcinomas, chronic achlorhydria, etc., and form one of our larger groups. Comparing them with those individuals in whom no such ailment is apparently present, we find no significant difference (see Table XII).

TABLE X—SYPHILIS.

	Males—Hearing Group						Females—Hearing Group					
Age	I	II	III	IV	V	Tot.	I	II	III	IV	V	Tot.
0-19							2					2
20-29	10	2		1	1	14	18	1			1	20
30-39	17	10	7	2		36	18	1	1		1	21
40-49	19	17	9	4		49	8	6				14
50-59	4	6	3	3	2	18	2	5	2	2		11
60-69			2	2		4		5	1			6
Total	50	35	21	12	3	121	48	18	4	2	2	74
Expected values on basis of cases without syphilis:												
	43.9	32.1	30.3	11.9	3.8	121	49.55	16.55	6.6	.5	1.8	74
	$X^2 = 4.13$		$P > .2, < .3$				$X^2 = 5.43$		$P > .2, < .3$			

TABLE XI—C.N.S. SYPHILIS.

	Males—Hearing Group					
Age	I	II	III	IV	V	Tot.
0-19						
20-29	3	1			1	5
30-39	4	5	4	2		15
40-49	2	7	3	3		15
50-59		1	2			3
60-69						
Total	9	14	9	5	1	38
Expected values on basis of cases without C.N.S. syphilis:						
	16.5	10.3	8.4	2.9	.9	38
		$X^2 = 6.31$	$P > .1, < .2$			

TABLE XII—GASTROINTESTINAL DISEASES.

	Males—Hearing Group					
Age	I	II	III	IV	V	Tot.
0-19	2	1				3
20-29	8	2				10
30-39	12	6	9	1	2	30
40-49	15	14	9	1		39
50-59	4	5	15	11	2	37
60-69	2	1	8	7	2	20
Total	43	29	41	20	6	139
Expected values on basis of cases without G.I. diseases:						
	41.3	36.5	36	18.3	6.9	139
		$X^2 = 2.58$	$P > .3, < .5$			

Carcinomas, Sarcomas, Tuberculosis: Malignant tumors together with tuberculosis represent the wasting diseases sufficiently represented in our material. That the nutritional balance may be of influence on hearing is quite possible, but here again our results are negative (see Tables XIII and XIV).

TABLE XIII—CARCINOMAS AND SARCOMAS.

	Males—Hearing Group						Females—Hearing Group					
Age	I	II	III	IV	V	Tot.	I	II	III	IV	V	Tot.
0-19							2					2
20-29	2	2				4	4					4
30-39	4	2	3			9	10	2	1			13
40-49	9	5	8	3		25	12	8		1		21
50-59	4	4	15	11	2	36	5	7	6			18
60-69	1	5	8	8	2	24		5				5
Total	20	18	34	22	4	98	33	22	7	1		63
Expected values on basis of cases without carcinomas:												
	20.4	25.1	29.7	15.6	7.2	98	35.94	17.52	6.97	.9	1.67	63
	$X^2 = 6.56$		$P > .1, < .2$				$X^2 = 3.05$		$P > .5, < .7$			

TABLE XIV—TUBERCULOSIS.

	Males—Hearing Group					
Age	I	II	III	IV	V	Tot.
0-19	3	1	1			5
20-29	12	3	1	1		17
30-39	4	1	2	1		8
40-49	2	4	2	1		9
50-59	1	2	3	2		8
60-69			3			3
Total	22	11	12	5		50
Expected values on basis of cases without tuberculosis:						
	22.8	11.95	8.65	4.1	1.5	50
		$X^2 = 2.95$	$P > .5, < .7$			

Goitres: Of the endocrine group a relatively sufficient number of individuals were the females with a diagnosis of goitre. (We could not, without dividing still more our numbers, separate these into toxic and nontoxic goitres.) Comparing these with our remaining population, we find again no difference (see Table XV).

We are forced to conclude from these analyses that in general none of the above systemic diseases cause more loss of high tones than any other disease, always of course within the limits of our material, which was far from sufficient. It must not be forgotten that there are many factors of which we know nothing and have accordingly been unable to take them into account. And these are mainly two: First, in no case did we know the length of time elapsed since the incidence of the disease. Second and principally, the constitutional factor, intending by this term the individual functional behavior

under the altered physiologic condition in question, whether this be general systemic or specifically otologic, this depending upon his inherent morphological habitus. It is known from general observation in otology, that under the same conditions, say occupation, some individuals suffer from impairment of hearing sooner than others. De Giovanni[17] has well observed: "In every morbid case it would be necessary to have present besides the theoreticopractical knowl-

TABLE XV—ENDOCRINE (GOITRES).

Females—Hearing Group						
Age	I	II	III	IV	V	Tot.
0-19	2	1				3
20-29	13	3			1	17
30-39	12	2				14
40-49	9	3	1			13
50-59	6	1				7
60-69	1		1			2
Total	43	10	2		1	56
Expected values on basis of cases without goitres:						
	38.9	11.8	3.7	.4	1.2	56
		$X^2 = 1.91$	$P > .7, < .8$			

edge of the malady, the knowledge of the circumstance by virtue of which it acquires special form and color, the knowledge of those special industrial attributes or characters that justify the inadequately interpreted axiom which says not diseases but individual patients must be treated."

SUMMARY.

1. The results of a statistical analysis of the hearing of 1,980 individuals examined with a Western Electric Co. 1-A audiometer, tuning forks, monochord, etc., are reported.

2. Special care was taken in the selection of the material and emphasis has been placed upon the method of analysis employed in seeking the interrelationships between the hearing and the various factors which may have influenced it, thus striving always to avoid drawing unwarranted conclusions.

3. Evidence is presented concerning the significance of the 4096 "dip" which is regarded as a first indication of the degenerative process which results in the loss of acuity for all high tones.

4. The analysis shows a significant and marked difference between the sexes in the degree of loss of acuity for high tones. Bunch and Raiford's observation in this regard is thus confirmed.

5. There is evidence indicating that middle ear inflammation may affect only the acuity for high tones; reasons are given for interpreting this as meaning that the inner ear has been involved.

6. The analyses made with respect to the possible influence of certain diseases, *viz.*, cardiovascular, syphilis, malignant tumors, gastrointestinal diseases, tuberculosis, goitres, show that individuals with a diagnosis or history of any of these diseases have no greater lowering of acuity for high tones than those without.

REFERENCES.

1. For a better understanding of the philosophy of statistics, its field of application and its limitations, cf. A. Niceforo, Il Metodo Statistico, Nuova Ed. Messina, 1932.

2. Discussion on D. W. Drury's "Aural Acuity and Brain Lesions, Audiometric Studies." *Trans. Amer. Otol. Soc.*, 1931.

3. DRURY, D. W.: Deafness in Syphilis; an Audiometric Study. *Ann. of Otol.*, etc., Vol. 38, p. 625, 1929.

4. A report of part of this study was presented by L. M. Polvogt at the meeting of the Americal Otological Society, May, 1932. Cf. Guild, Crowe, Bunch and Polvogt, Correlations of Differences, etc., *Acta Oto-Laryngologica*, Vol. 15, p. 269, 1931.

5. POLVOGT, L. M.: Loc. cit.

6. ZWAARDEMAKER, H.: Der Verlust an hohen Tönen mit zunehmenden Alter. *Arch. f. Ohrenheilk.*, Bd. 32, p. 53, 1891.

7. STRUYCKEN, H. J. L.: Tabellen über die obere Hörgrenze bei pathologischen Verhältnissen. *Passow's Beiträge*, Bd. 6, p. 289, 1913

8. BUNCH, C. C.: Age Variations in Auditory Acuity. *Arch. of Otolaryng.*, Vol. 9, p. 289, 1929. Further Observations on Age Variations in Auditory Acuity. *Ibid.*, Vol. 13, p. 170, 1931.

9. BUNCH, C. C., and RAIFORD, T. S.: Race and Sex Variations in Auditory Acuity. *Arch. of Otolaryng.*, Vol. 13, p. 423, 1931.

10. See the various articles by Pearson, *et al.*, beginning with: On the criterion that a given system of deviations in the case of a correlated system of variables is such that it can reasonably be supposed to have arisen from random sampling. *Phil. Mag.*, p. 151, 1900. The medical man can obtain useful knowledge of the significance and of the method of calculation of the more commonly used formulae in R. Pearl's Introduction to Medical Biometry and Statistics, 2nd Ed., Phila., 1930.

11. FISHER, R. A.: Statistical Methods for Research Workers. London, 1928.

12. STRUYCKEN, H. J. L.: Loc. cit.

13. POLITZER, A.: A textbook of the diseases of the ear and adjacent organs. English translation from the 3rd German edition, by O. Dodd.

14. WANNER, F.: Functionsprüfungen bei acuten Mittelohrentzündungen. *Zeitschr. f. Ohrenheilk.*, Bd. 43, S. 61, 1903.

15. STRUYCKEN, H. J. L.: Loc. cit.

16. BUNCH, C. C.: Further observations, etc., loc. cit.

17. DE GIOVANNI, A.: Clinical Commentaries Deduced from the Morphology of the Human Body. Translated from the 2nd Italian edition by J. J. Eyre, New York, 1910.

CYTOLOGICAL AND BACTERIOLOGICAL STUDIES OF OTITIC DISEASE.*

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During the last year we have interested ourselves in various fundamental problems of otolaryngology. The viscosity, the pH, the nitrogen content, and the CO_2 content of the secretions have been studied. By interfering with calcium metabolism we have produced an experimental eosinophilia followed by infection of the membrane. The influence of acidosis and alkalosis on the eosinophilia has been studied.

Secretory nerves have been stimulated to note the effect on the mucosa; also the influence of various drugs have been observed. We have produced an experimental allergy. The work in physics, in anatomy, and in the pathology of the ear has been continued.

These problems are complicated and the results secured have more influence on nasal sinus disease than on the ear. Practical results came from the study of the cytology of the discharges.

In our work in bacteriology of the ear, we have not concerned ourselves so much with the routine study of all cases, but rather have made complete studies of groups of interesting cases.

The hemolytic streptococcus and the hemolytic staphylococcus have been the outstanding etiological factors in the cases of acute otitis. The importance of securing the material for bacteriological examination at the time of myringotomy and at the time of mastoid operation has been repeatedly emphasized.

The Pneumococcus Type III has often been found at the time of myringotomy. It has been absent on repeated examinations of the ear discharge and was then found again at the time of operation on the mastoid.

The normal tympanic cavity we have found to be usually sterile and if cultures are made at the time of myringotomy, we will probably pick up the etiological factor. Following myringotomy there is an invasion of contaminating organisms and it is almost impossible to say what the significance of an organism is if one is found. In many of our cases the true etiological factor has been lost in the mass of contaminants.

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All cases studied were examined for both aerobic and anaerobic organisms. Most anaerobic organisms are secondary invaders; they have much to do with the production of the foul odor in the ear. The foul odor is due to the presence of incompletely oxidized cleavage products. These organisms are found to be more abundant in destructive processes. Ordinarily they are harmless, but they may contribute to a pathological process under suitable conditions. They cannot be disregarded as important factors. For instance, in one case of mastoiditis with thrombophlebitis, abscess in the neck, septicemia and finally meningitis, the bacillus melaninogenicum was found and was the only organism found in the neck, in the blood stream, and in the spinal fluid. If it was present in the ear itself at the beginning of the infection, its presence was not detected. Certainly in the last two weeks of the patient's illness, a most careful search failed to reveal any other organism than the bacillus melaninogenicum, and it seemed to be the only etiological factor of the septicemia and the meningitis. When the neck became infected, it was the only organism found. When meningitis developed, it was the only organism present in repeated examinations of the spinal fluid. With the development of septicemia it appeared in the blood. In a second case of otorrhea this organism was found, but in this instance it had little clinical significance.

It is conceivable that we have not been searching for anaerobic organisms as we should. That we have not been giving the consideration to the secondary invaders that they deserve.

The pathological process so changes resistance that the bacillus melaninogenicum becomes an etiological factor. This process is not so different from that reported by Shurley where deficient diets resulted in nonpathological organisms becoming pathological.

We must remember that the factors influencing infection of other parts of the upper respiratory tract influence the ear; that the organism concerned and its virulence is only a small part of the picture. The problem of resistance to infection is as important in the ear as elsewhere.

The formation of serous discharge in early otitis is the first defense against infection. It is the result of irritation and the excess of fluid is to get rid of the irritant. At first this secretion has a mild bactericidal action. Some time ago Mr. Linton showed that this was soon lost. However, this current of secretion plus its bactericidal action sometimes overcomes the invasion by the bacteria.

In the event the irritant is not washed away, but reaches the cell, I am inclined to think that the next defense is the desquamation of

groups of superficial epithelial cells. These cells are thrown off in small masses, carrying the bacteria with them.

If we spray a virulent streptococcus into the nose of a rabbit; if it is so active that it will cause septicemia and death in 24 hours, at autopsy the membrane shows very little change. The only marked change is the distension of all the goblet cells. Apparently an earnest effort is being made to wash away the irritant. The ciliated cells are normal with intact cilia; there is no subepithelial infiltration of cells. The method of penetration by micro-organisms has never been definitely determined.

If a less virulent organism is used the next change noticed is the death of and exfoliation of groups of epithelial cells. In one series of animals streptococci are seen in cells. The next change is a dense infiltration of the subepithelial layer with mononuclear cells. Later the polymorphonuclear cells come into the picture.

If before the animal is injected a vaccine is used in the nose there is a great increase in the number of mononucleated cells present.

The Pneumococcus Type III has continued to be a very dangerous organism. We have found it often at the time of mastoidectomy when its presence was not suspected, owing to the fact that it could not be picked up in the ear discharge. These cases have all behaved as the routine infections of the mastoid do and the finding of the organism at operation does not in any way influence our prognosis. The finding of this organism at the time of myringotomy does have a decided bearing on our prognosis. In every case in which this organism has been found it has been necessary sooner or later to do a mastoid operation. One should exercise the very best clinical judgment. If the blood count, the temperature and the general condition of the patient will allow it, he should postpone his mastoid operation until Nature has had an opportunity to limit the infection. Early operation is to be avoided if possible. The fact that in all cases where the presence of this organism was not suspected but was found at operation did so well demonstrates this. We should not do a mastoid operation as soon as the Type III pneumococcus is discovered. We should realize that in almost every case, sooner or later, a mastoidectomy will be needed. A conservative course gives the best results.

The prognostic significance of Pneumococcus Type III was nicely illustrated in the case of one infant, age about 1 year. This infant developed an acute otitis of mild severity. In a few days there was no fever, no leukocytosis and only a very slight discharge from the ear. This infant at myringotomy showed the presence of Pneu-

mococcus Type III. The only reason for not sending the infant out of the hospital and treating it at home was our fear of this organism. In the course of eight or ten days, when the child was in good condition except for the very slight ear discharge, a slight fever developed, a mastoidectomy was performed. The child developed a lung complication and diarrhea with bloody stools, and died. Here was an infant healthy, except for the slight ear discharge of seven or eight days duration. We realized the exigencies of the situation, but in spite of what seemed to us to be the very best pediatric and otologic treatment, the child died. The child was not very ill until several days following the mastoid operation. The operation seemed to have little significance.

We have not had during the year one proven case of tuberculous otitis. We have had two cases, one with tuberculosis of the kidney, the other with pulmonary tuberculosis, where the bacillus tuberculosis was found in the ear discharge and injection of guinea pigs gave positive results. The usual number of cases with organisms in the discharge resembling bacillus tuberculosis have been found.

Many years ago I investigated a large series of cases of chronic otorrhea in cases with pulmonary tuberculosis and was unable to find evidence in the tissue or bacteriological evidence of tuberculosis of the ear in all the studied cases. The proven cases of tuberculosis in my experience had a bad result whether they were operated or not. The infection is through the blood stream and the pathology is that of bone tuberculosis.

In a case of maxillary sinusitis, we secured the bacillus tuberculosis from the chronic discharge from the sinus and our injection of guinea pigs with this discharge gave positive results. The sinus was later operated and the opinion of the pathologist was that there was no indication of tuberculosis of the lining of this chronically infected sinus. This patient had a quiescent pulmonary tuberculosis and a good result so far as the sinus was concerned was secured from the operation. Laboratory investigations showed it to be nontuberculous. The two cases that we have had during the year with bacillus tuberculosis in the ear discharge have also had present in the ear the hemolytic streptococcus. They began with fever and pain, as do those with streptococcic infection. One required a mastoidectomy, the other healed following myringotomy.

When an individual with a tuberculous lesion develops an infection in any other part of the body, the bacillus tuberculosis is very likely to invade the tissue as a contaminant and the finding of the organisms does not necessarily indicate that the lesion is tuberculous.

Clinically there was nothing about our two cases to suggest that they were tuberculous. The presence of bacillus tuberculosis in the ear discharge as proven by the guinea pig inoculation does not indicate tuberculosis of the ear, if the patient has an active tuberculous lesion in some other part of the body.

In connection with the cytological examination of the ear discharge, in one case yeast cells were found in the ear discharge in great abundance. The patient was an infant suffering from malnutrition.

Torulae are found in the throats of poorly nourished infants. They may occur in the external auditory canal in any case. When present in the throats of infants or in the ear canals, they have little or no clinical significance. This ear infection was not clinically a case of coccidiosis. The ear was well in a few days. The yeast cells were undoubtedly contaminants and had no significance.

Cholesterol: In making cytological studies of ear discharges, we became interested in the frequency with which cholesterol crystals plus fatty degenerated cells were found. These two things were found present many times more frequently than ever before. The reason for this is that in studying the cells in the washings from the ears the material was centrifuged and the precipitate examined. The cholesterol crystals were found in the precipitate.

Cholesteatoma is said to be definitely diagnosed by the presence of cholesterol crystals plus fatty degenerated cells in the ear discharge. Some authors feel that the presence of cholesterol crystals alone is pathogomonic of cholesteatoma (Rejto, *Arch. Otolaryngol.*, 13, 1931).

We have found the crystals plus the fatty degenerated epithelial cells in so many cases that we are inclined to doubt the significance of the findings. Certainly, clinically, one would not advise radical operation on some of the patients that had the crystals in the ear discharge. The crystals of cholesterol were present in the discharges in a very much larger percentage of cases of chronic otorrhea than we find cholesteatoma when operation is performed. The fact that we find cholesterol and fatty degenerated epithelial cells in the discharge present in the external auditory canal does not necessarily mean that the cholesterol and cells are coming from the middle ear.

It seemed advisable to ascertain whether cholesterol in the ear discharge was peculiar to cholesteatoma. Using the acetic anhydride test or the sulphuric acid test, together with the polariscope, certain studies were made.

In cerumen there was found by the colorimetric test, a very high percentage of cholesterol. The color reaction was more marked than

we have secured from any ear discharge. The cerumen is densely impregnated with crystals. They do not have the characteristic form of cholesterol. They are, however, probably cholesterol. The uncertainty is sufficient to cast doubt on the significance of cholesterol crystals in any discharge found in the external auditory canal. Cholesterol was found in the ear discharge of acute otitis. The older the discharge the greater the amount as a rule.

It was quite evident that the presence of cholesterol was not peculiar to cholesteatoma.

After we had begun our work we found that Rejto (*Arch. Otolaryngol.*, 13, 1931) had made a study of the colorimetric method of determining the amount of cholesterol. He felt that a very strong color reaction was diagnostic of cholesteatoma and that a weak reaction excluded it. Salkowski, according to Rejto, is of the opinion that the cholesterol in cholesteatoma is not always crystalline. He estimates that there is from 18 to 23 per cent of cholesterol in cholesteatoma, and 10.6 per cent in normal epidermis. Lautenschlager believes the cholesterol in cholesteatoma causes bone absorption. With the polariscope crystals can be found in the purulent discharge of recent cases of acute otitis. While they are not the characteristic cholesterol crystals, they do not differ from the crystals sometimes found in known cases of cholesteatoma.

But little is known of the uses of cholesterol. It is a protective agent. It combines with toxic substances.

In sections of temporal bone with mastoiditis we often find diamond-shaped spaces in granulation tissue which contained cholesterol before it was dissolved out by the solutions used. There is no suspicion of cholesteatoma in or near these masses so that in chronic suppurative otitis, cholesterol in the ear is certainly not peculiar to cholesteatoma. As yet we have not determined the percentage of cholesterol in granulation tissue in the mastoid.

Because of the clinical significance of cholesteatoma and because of the diagnostic significance of cholesterol in the ear discharge, it seemed desirable to determine just what amount of cholesterol was present in the ear discharge. Cholesterol is insoluble in water. Even if some such substance as carbon tetrachlorid, in which it is soluble, is used the location of the cholesteatoma or cholesterol in granulation tissue would determine the contact which the drug would have with the cholesterol. It does not seem that the same opportunity for the cholesterol to get into the ear discharge would always be present.

Following the procedures used by Elman and Taussig (*Journal of Laboratory and Clinical Medicine*, Dec., 1931) on the quantitative

determination of cholesterol in bile, two methods were used. The polariscope was used to determine the presence of crystals and the colorimetric method for quantitative chemical work.

Colorimetric determinations are made as follows:

The ear washings are evaporated to dryness over a water bath; the residue saponified with 3 per cent KOH in 95 per cent alcohol for 20 to 30 minutes (over a water bath).

The cholesterol is extracted from the alcohol with petroleum ether in a separatory funnel, layers separated. The petroleum ether is evaporated; and the residue extracted with chloroform.

Standard cholesterol solutions of different strengths are prepared in order to have one containing approximately the same amount of cholesterol as the unknown.

The unknown is made up to exactly 10 c.c. with chloroform. Ten c.c. of each of the known standards are pipetted into dry test tubes. Two c.c. of acetic anhydrid plus 0.2 c.c. of concentrated sulphuric acid is added to each tube (known and unknown) and all of the tubes subjected to the same light. The color is allowed to develop for about 15 minutes. By comparing the unknown with the known closest to the color of the unknown, in the colorimeter, the amount of cholesterol in the sample is determined.

Certain factors influence this test. Time, temperature and light influence the color. The method is simple and under uniform conditions gives accurate results.

In every case examined crystals of some kind have been found by using the polariscope. Every middle ear discharge has given a colorimetric reaction of cholesterol. The subject which is being investigated is a very complicated one and it will be a long time before we, at least, will be able to express a definite opinion as to the actual significance of the presence of or the absence of cholesterol crystals as a diagnostic factor in cholesteatoma and the significance of the presence of large amounts of cholesterol as a diagnostic point.

We believe that the presence in the ear discharge of cholesterol crystals with fatty degenerated epithelial cells does not positively indicate the presence of cholesteatoma, neither is it indicated by a very marked color reaction for cholesterol.

Washington University Medical School.

MASTOID ROENTGEN PATHOLOGY.*

Dr. Louis J. Gelber, Newark.

In order for one to have a thorough understanding of Roentgen mastoid pathology, it is essential to know the normal anatomy of the mastoids from birth on to adolescence. This is necessary because of the character of the changes that progress in this bone from infancy on. The mastoids are situated at the sides and base of the skull, each one forming one-fifth of either temporal bone. The remaining four-fifths are made up of four components: namely, squama, petrous, styloid and tympanic parts.

For one to clearly interpret pathology by Roentgen rays, we must understand the relation of one structure to the other. Therefore, for our purpose and in order to fully comprehend this treatise it is essential to know that the mastoid cells are the most superficial structures and form our outer layer. Deeper to this is our sigmoid sinus. Slightly deeper to this is our sinus plate. As we proceed further internally we come upon semicircular canal and internal ear and finally the posterior part of the middle fossa of the skull. It is also important to know that the antrum lies somewhat superficially in close relation to the mastoid cells and immediately below the tegmen tympani. The latter is a thin plate of bone that separates the brain from the antrum and mastoid cells. Superficial to the antrum is the squama, while deep and internal to the antrum is the lateral semicircular canal also lying beneath the tegmen but occupying the petrous portion of the temporal bone.

Therefore the mastoid cells and antrum are superficial to the sigmoid sinus and separated from one another by a very thin lamina of bone. Deep to this is the semicircular canals lodged in the petrous portion of the temporal bone.

Having therefore obtained a clear relationship of the structures in the mastoid region, we must proceed to learn something about the early development of these structures and the relation that one bears to the other from birth to adolescence.

At birth the temporal bone can usually be separated into its various component parts. The lateral surface of the petrous portion forms the inner side of the tympanic antrum. The antrum is very prominent in infants from birth to 6 months of age. It is the only pneumatic space present in the newborn. As yet the mastoid process

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is undeveloped and it is not until the second year that it assumes a nipple-like form. From this time on the spongy substance of the mastoid process becomes permeated with air cells which seem to be diverticular extensions from the antrum and communicate with the antrum. Of course, pneumatization of the mastoid may fail to occur and remain in its original diploic state. Therefore we observe that at birth the various components of the temporal bone (the organs having to do with hearing and equilibration) come first, whereas the mastoid process with its air-containing pneumatic structures come last.

Evans[1] seems to believe that "pneumatic mastoid structures are frequently observed before the end of the first year of life, whereas the adult mastoid structure can be observed as early as the second year."

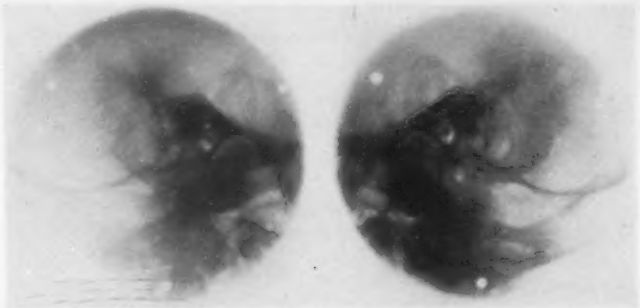

Fig 1. Shows the diploic type of mastoids with innumerable small septal divisions.

Schwartz[2], on the other hand, quotes such authorities as Kopetzky and Politzer in stating that at birth the mastoid consists of a single cell, the mastoid antrum, which already has reached its full size.

Authorities such as Zucherkandl[3] claim that the mastoid process reaches its adult form in the third year of life. Dr. Law[4], on the other hand, has observed pneumatic structure as early as six months.

Therefore, in discussing mastoid Roentgen pathology we can see how important it is to know the exact age of our patient because the sigmoid sinus is an important guide towards the existence of any pathology below 2 years of age. Granger[5] claims that in normal conditions and under the complete absence of pathology, the sigmoid sinuses are not visible before pneumatization has occurred (under two) and if seen before this age period, pathology should be suspected. Besides, the sinus does not show distinctly in normal mastoids. However, when mastoids become involved the lateral sinus

becomes prominent. Therefore, in radiography of mastoids of children under two with running ears, whenever we see the mastoid processes without cells we must think of active destruction going on.

Mastoid cells may early assume one of three types, according to the size and number of cells present. These three types are *a.* diploic, *b.* sclerotic, *c.* pneumatic.

a. In the diploic type, numerous small cells are scattered throughout the mastoid process. These cells are minute in size and may number several hundred. A persistence of this type is indicative of failure of pneumatization.

In infancy the mastoids are nothing more than diploetic structures with the exception of the mastoid antrum and a few cells on its outer wall. This infantile, diploetic type structure appears to persist in 15 per cent of people throughout life. Sclerosis occurring around the antrum in these infantile cases appears to be the result of superimposed chronic suppurative conditions. At times it seems almost impossible to differentiate between a sclerosed mastoid and a diploetic one, Roentgenologically.

In the diploetic mastoid we can usually distinguish: *1.* Tegmen; *2.* tip of mastoid process; *3.* auditory canal; *4.* anterior wall of sinus; *5.* extremely fine meshwork in triangular area.

b. In the sclerotic type, the cells are absent and the process merely consists of osseous diploe or dense bone. This triangular dense bone is limited posteriorly to the lateral sinus groove, superiorly by the tegmen, and anteriorly by the posterior canal wall. This type is usually the result of new bone deposit replacing and obliterating previously existing cells.

In the sclerotic mastoid we can usually distinguish: *1.* Well marked anterior sinus groove; *2.* tegmen; *3.* usually associated with a chronic process in mastoid with oft repeated attaches of otitis.

c. In the *pneumatic type* the whole of the process becomes excavated by few but large cells. The sigmoid portion of the transverse sinus is in immediate relation with the medial surface of the mastoid process.

In the pneumatic mastoid we can usually distinguish: *1.* Small cells in triangular area; *2.* large cells in tip of mastoid and posterior to sinus groove.

We can therefore readily see that the Roentgenogram of the mastoid regions serves well as an anatomical guide in furnishing information as to the size and type of the mastoid cells as well as the position and course of the lateral sinus.

Roentgen examination of the mastoid process depends upon the presence of air-containing cells in the bony structures. Because of

the wide variations of the normal mastoids as above described, it is customary, as well as essential, to make films of both mastoids when pathology is suspected. No attempt at any time should be made to interpret films of the mastoid regions unless both mastoids are taken. This is quite evident as the fundamental basis for reaching a diagnosis is a comparison of the affected with the unaffected sides.

How are radiographic interpretations made in pathologic mastoid conditions? To answer this question is to remember one thing;

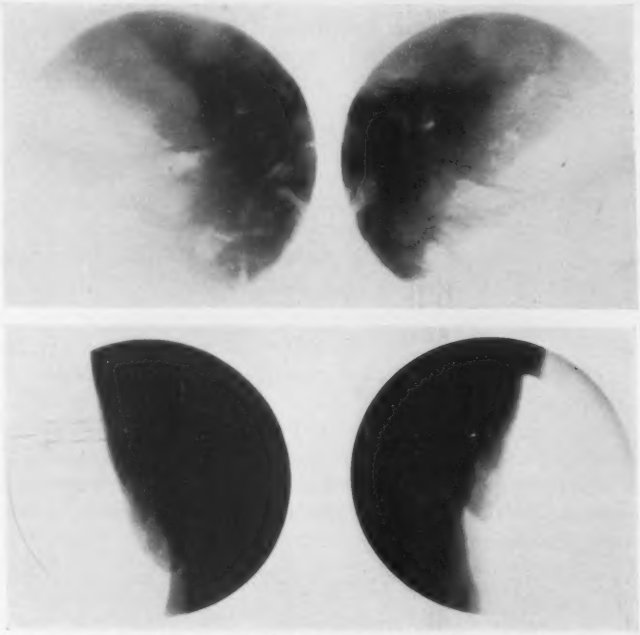

Fig. 2. Shows sclerotic mastoids in a patient 25 years of age. In all probabilities the result of a superimposed infectious condition.

namely, that X-rays produce varying shadow densities in going through structures of different specific gravity, as bone, muscle, fluid, etc. Furthermore, that pathological conditions in the mastoids result in varied structural densities that affect absorption of the X-rays.

Fortunately, the mastoid regions contain air-containing cells and a close study of the air contents of either mastoid area is taken advantage of in Roentgen diagnosis and pathology of their struc-

ture. Therefore, mastoid Roentgen diagnosis depends upon *a.* the air content, *b.* the condition of the bony septa.

Any well aerated space in the body absorbs little or no X-rays. Whether our area under examination are the lungs, sinuses or mastoids, the X-rays will pass through these air-containing cavities with the minimum of absorption. However, as soon as our specific gravity or density of the structures increases either by fluid or products of inflammation, this decrease in aeration immediately results in a greater amount of X-ray absorption, which becomes evident on our X-ray films. Therefore, the pathological mastoid will vary in its illumination from a very slight decrease to a total absence of aeration, depending on whether a small amount or a great amount of inflammation exists. Furthermore, as the products of inflammation collect in the mastoid cells, the specific gravity increases and our radioparency also becomes less because of the increase of density.

We next focus our attention on the bony septal division. With the products of inflammation increasing and more destruction ensuing, changes in the bony septa result from the apparent increase in structural density, resulting in osteoporosis and finally destruction of the thin bony walls.

The otologist may state, and correctly so, that he is not interested in X-ray absorption or nonabsorption and wishes an explanation of the Roentgenologist's findings in terms of pathology. The only enlightenment that the X-ray man has to offer to this otologist is that the findings of pus or serum may compare to the Roentgenologist's report of decreased illumination due to air displacement by the products of inflammation. Where septa have been reported absent by the X-ray man, the otologist might find complete bony necrosis and destruction.

In a standard X-ray projection the following landmarks should be recognized:

a. The external auditory meatus deep to which is the internal meatus.

b. The mastoid antrum which is located superior and posterior to the external auditory meatus.

c. Sigmoid sinus directly posterior to meatus.

d. Tegmen located above antrum.

e. Temporomandibular articulation in front of external auditory meatus.

f. Emissary vein between knee and tip of mastoid.

g. Triangular area is that part bounded by tegmen above auditory meatus anteriorly and anterior wall of sinus posteriorly.

Findings in Acute Mastoiditis: 1. Slight veiling of involved side. *2*. Distinct cell trabeculae. *3*. Seen in about 50 per cent of cases with simple inflammatory otitis media.

Findings in Acute Purulent Mastoiditis: 1. Marked clouding of involved side obscuring antrum. *2*. Areas of septal necrosis may or may not be present.

Chronic Purulent Mastoiditis: 1. Marked veiling of mastoid cells. *2*. Necrosis or thickening of septal division coalescence of cell walls. *3*. Blurring of tegmen and anterior sinus groove, with possible extradural and perisinus abscesses.

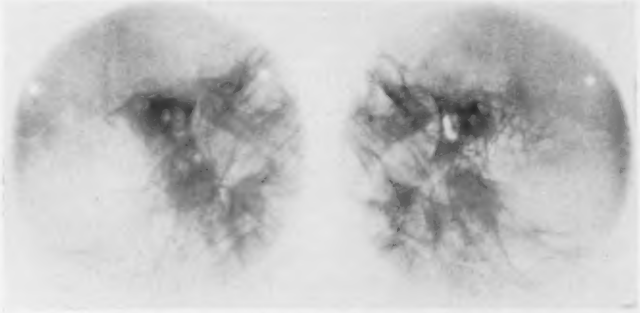

Fig. 3. Shows the cells on the right side fairly large, those on the left faintly visualized. A beginning necrosis and destruction of the septal walls on the left side.

To Summarize:. Normal mastoids are alike in the same individual. X-rays of mastoids may tell us anatomical relations as well as pathological involvements.

Mastoid cells may be of diploic type which never develop. They may be of cellular type, which may be large, medium, small or mixed. The sclerotic type may be present, which means that disease has previously existed.

Early in the pathology of mastoiditis a peppering of the cells may exist. After a few days, examination of the affected mastoid region appears hazy and Roentgenography of this area appears as though the region were out of focus. As the disease progresses, septal necrosis and coalescence formation become apparent.

Finally, the greater amount the destruction, the more clearly the lateral sinus is apt to show and we then begin to get a clear view of the lateral sinus until exposure of the sinus. If our destructive process continues, our sinus plate becomes involved and the lateral sinus

takes on a hazy appearance with probable resultant perisinus abscesses or lateral sinus thrombosis.

We conclude, therefore, in stating that radiography of the mastoids is nothing more than a *study of density contrasts.*

BIBLIOGRAPHY.

1. EVANS, WILLIAM A.: The Value of Roentgen Study of Mastoid Diseases in Children Under Five. *Amer. Jour. Roent. and Rad. Ther.*, 1923, pp. 382-385.
2. SCHWARTZ, L. H.: Is Mastoidectomy Necessary in Young Children? *Arch. Otolaryng.*, 1927, pp. 353-360.
3. ZUCKERHANDL, E.: *Handb. I. Obrenbeil.*, pp. 33-34.
4. LAW, DR.: Personnel Discussion.
5. GRANGER, AMEDEE: *Radiology*, p. 495.

41 Lincoln Avenue.

MODIFICATION OF THE LAUTENSCHLAEGER OPERATION FOR THE SURGICAL TREATMENT OF OZENA.*

DR. ALFRED WACHSBERGER, New York.

M. D., female, white, age 22 years, Italian nationality. *Past History:* The outstanding facts in the past history are measles and scarlet fever in early childhood, pneumonia twice, at the ages of 7 and 16 years, and what she termed as a nervous breakdown at the age of 19 years. *Family History:* Family history is entirely negative for symptoms referable to atrophic rhinitis. *Present History:* When she first came to me, on Dec. 4, 1930, she complained of clogging of the nasal passages, a purulent discharge and crusts from the nose. There was a bad odor, which was noted objectively as well as subjectively. She also had bilateral frontal and infra-orbital headaches. All of the symptoms started at puberty and became progressively worse up to the time of examination.

Examination: The nose revealed the usual picture of ozena. The nasal cavity and the nasopharynx were lined with crusts. There was an atrophic pharyngitis and a chronic laryngitis. The middle turbinates were also atrophic to a moderate degree. No pus could be seen exuding from any of the sinus openings. Blood Wassermann was negative. The cholesterol content of the blood serum was 134 m.gm. per 100 c.c. before the operation, and 170.6 m.gm. one month after the operation. After the nasal cavity was cleaned of

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its crusts, the inferior turbinates were seen to be reduced to mere ridges. Radiographic examination showed both antra to be small but air-containing. The walls were sclerotic.

Operation: On Dec. 27, 1930, the patient was operated on in Polyclinic Hospital under local anesthesia. After injection of both sphenopalatine ganglia, I performed my modification of the Halle-Lautenschlaeger operation to narrow the nasal cavity. An incision was made in the nasal vestibule on the pyriform crest, and through this incision the bony facial antral wall was exposed and the mucoperiosteum separated from the nasoantral wall. The antrum was opened at the facial wall with a chisel and one blade of a Killian speculum introduced through this opening, while the other blade was placed between the mucoperiosteum and the bony nasoantral wall. The latter appeared between the two blades of the speculum, as the septal cartilage does in a submucous resection. The inferior attachment of the nasoantral wall was severed along the floor of the nose with a chisel, special bone scissors and a saw. After indentation of the pyriform crest up above, the lateral wall was fractured toward the septum. It was held in place by packing the antra firmly with iodoform gauze. This procedure was repeated on the other side.

After-Treatment: The packing was removed after five days and the antra repacked. This was repeated twice at the same interval, at the end of which time firm adhesions between the turbinates and the septum had been established.

The advantages of this modification over the Halle-Lautenschlaeger method are as follows:

1. The whole procedure is done submucously, which prevents a permanent opening from the nose to the antrum.

2. It is practicable in advanced cases because more of the wall is mobilized and a greater narrowing of the nasal cavity is obtained.

3. The size of the antrum does not affect the outcome to the same extent as with the other methods.

4. It is easier to perform, technically, and the after-treatment is simpler.

Result: To judge the final result of the operation the patient will have to be under observation for a longer period of time. My longest observations of my modification of this operation is about one year. The patient I am presenting is relieved from all her symptoms. There is no crust formation and the fetor is gone entirely. When I will have operated a sufficient number of cases I shall report the results in a more detailed manner.

51 West 73rd Street.

NASOPHARYNGEAL FIBROMA; REPORT OF TWO CASES.*

DR. WILLIAM SPIELBERG, New York.

A considerable number of cases of nasopharyngeal fibroma have already been reported in literature in this country. We feel, however, that due to the infrequent and even rare occurrence of this condition, the addition of our two cases will be of some value. We, furthermore, feel that the general character of the cases to be reported and their complete workup (*i. e.*, history, physical findings, photographic reproduction of the tumor and complete pathologic studies followed by operative treatments, and postoperative results) will add as well considerable value to the study of this interesting condition.

Personal inquiries made from otolaryngologists reveal that many have never seen a case of nasopharyngeal fibroma, while some have seen only a case or two, either in their private or hospital practice.

Two cases reported by Dr. Persky from the private practice of Dr. Ersner, of Philadelphia, are interesting. Both occurred within one month. Boys of 10 and 15 years old, respectively. Both were operated on under general anesthesia and the tumor removed by snare. They encountered considerable bleeding in one case; and used radium in the postoperative treatment of both. Their cases recovered with no recurrence.

Coates reported a case of true fibroma in 1917. Others who reported cases and their observation on origin, growth, pathology and treatment are Goldsmith, Hall, Tilly, Getchell and Ucherman. New and Figi, of the Mayo Clinic, report an interesting series of cases.

A brief summary of the observations made by the above named authors may be made as follows:

1. *Age:* Nasopharyngeal fibromata occur between the ages of 10 and 25 years.

2. *Sex:* It appears to be predominant among males. New and Figi, as well as Getchell, report its occurrence in females.

3. *Origin:* The tumor is usually single, and rarely multiple. Arises most frequently in the nasopharyngeal recess. Other locations may be from the diseased fibrous tissue of the nasal process of the occipi-

*From the Otolaryngological Department of Beth Israel Hospital.

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tal bone, the anterior sphenoidal wall and sphenopalatine wall of nasopharynx, around mouth of Eustachian tube, posterior surface of palate, pterygomaxillary recess.

4. Pathology: The tumor may be a pure fibroma, polypoid fibroma or fibromatous polyp, depending largely upon where it originates. Microscopically, it is composed of fibrous tissue with large cavernous blood spaces, particularly at its attachment. It grows by extension and invades the orbit, accessory sinuses, pterygoid and temporal fossae or intracranial cavity. The tumor derives additional blood supply from the soft parts to which it becomes attached.

5. Treatment: Various forms of treatment, both surgical and non-surgical have been used, among which were caustics, electrolysis and galvanocautery. The snare was most frequently used, particularly in pedunculated tumors. When tumor had a broad base, avulsion was used and tumor torn from its attachment by a slow rocking motion of the instrument used. Radical surgical procedures such as external operation and splitting of the palate have been condemned. Radium both as salt and emanation have been used, both pre- and post-operative, by Milligan, Dahney, Crow, Baylor and Keith. New and Figi describe three methods they have used in radium treatments of nasopharyngeal fibroma. *1.* A capsule containing 50 m.g of radium applied into the nares for 10-15 hours. *2.* Steel points containing radium emanation inserted into the tumor. *3.* Radium inserted in the tumor. Treatments were repeated in six weeks to two months. Cases received two to nine treatments. X-ray radiation has been used by some investigators in addition to radium therapy, with report of a great many cases without recurrences.

CASE REPORTS.

Case 1: History: A. P., male, age 14 years, well developed and nourished. Appears in good physical condition. Came to my office for examination, June 19, 1930. Had tonsils and adenoids removed when 6 years old. Had usual diseases of childhood and does not remember to have ever been seriously ill. For several months has been suffering with rather distressed nasal obstruction, mostly on the left side, particularly at night. For the past two months has been getting frequent attacks of epistaxis. Gets very frequent nasal colds and discharge. The most aggravating symptom, however, is the nasal obstruction for which he came to seek relief.

Rhinoscopic examination revealed a moderate grade of nasal atrophy with purulent secretion filling both nares. A large mass was found filling the posterior two-thirds of the left nasal cavity extending both upwards in the nose and nasopharynx and somewhat down

below the soft palate into the aurophary nx. After swabbing the nose with cocain adrenalin solution, it was found to be very hard in consistency and firmly attached to the anterior sphenoid wall and lower nasopharynx by a wide base. The mass was completely filling up and obstructing the post nares so that the patient was unable to breathe through the left side of the nose. On forceful inhalation some air was admitted, but none could be exhaled due to the ball valve action of the tumor against the posterior nares and soft palate.

Throat: Postnasal drip of mucopus. Larynx and ears were negative. *X-ray examination* of the nasal accessory sinuses revealed chronic hyperplastic sinusitis with diminished aeration of the frontals, ethmoids, antra and sphenoids.

Diagnosis: A diagnosis of nasopharyngeal fibroma and chronic suppurative sinusitis of the atrophic type was made.

Several pre-operative treatments of argyrol tampons and including two treatments with zinc ionization to tumor mass were given without any relief to patient.

Operation: On July 16, 1930, the patient was admitted to the Beth Israel Hospital and the nasopharyngeal tumor removed. It is the preference of the author whenever possible to operate under local anesthesia. By this means a bloodless and painless field is obtained and operation can be performed under direct vision.

Procedure: The nasal cavity and tumor were packed with pledgets of cotton moistened in cocain adrenalin solution (10 per cent and 1-1000, respectively, equal parts) and left *in situ* for 15 minutes. The packing was then removed, and by means of a long thin needle the tumor mass was thoroughly injected with one-half per cent novocain adrenalized (8-10 minims of 1-1000 adrenalin to one ounce of novocain solution). By means of an extra heavy Greenwald forceps passed into the left nasal cavity, the center of the tumor mass was grasped and by a slow rocking motion, avulsed from its attachment and removed *en masse* through the nose.

There was no pain and no bleeding. The posterior nares were packed with iodoform vaseline gauze, pushing it posteriorly tight up against the nasopharyngeal wall. The packing was left in place for five days and removed. There was no postoperative bleeding and none whatever after removal of packing. Last examination, on Sept. 5, 1930, patient feels well and no evidence of recurrence.

Case 2: History: F. C., female, age 23 years, first examination Oct. 22, 1926. Nasal obstruction for one year with inability to breathe through left nostril. Profuse purulent discharge from both nares. No headaches. No general complaint.

Findings and Operation: A diagnosis of nasal polypi was made at that time and left ethmoidectomy performed on Oct. 26. On comp'eting the operation, a large mass was seen occupying the left nasopharynx.

At a subsequent operation, Nov. 2 (one week postoperative), the nasopharyngeal tumor was found to be attached to the vault of the nasopharynx. On November 16, a large pear-shaped growth of hard consistency was removed from the nasopharynx with a Brandagee forceps by grasping the growth behind the soft palate through the mouth and by a slight twisting motion removed *in toto* with peduncle attached.

Subsequent examination on Jan. 19, 1927, and July 2, 1929. No recurrence and patient free from other disturbing symptoms she had prior to the operations.

This case occurred in Dr. Kopetzky's private practice and was operated on by Dr. Almour, to whom I am grateful for permission to report this very unusual and interesting case.

Pathological report by Dr. Alfred Plaut, Director of Laboratories at the Beth Israel Hospital:

Case 1: Tumor from nasopharynx from Patient A. P., admission No. 35904; Path. No. 18083.

Microscopic: Dense fibroma with many wide spaces which are lined with endothelium. The connective tissue is formed by thick, wavy bands which stain heavily with eosin. Most of the nuclei are irregularly ovoid, some of them slightly star-shaped. Very few spindle-shaped nuclei are seen outside the wall of the vessels. At a few points small round cells are accumulated around vessels. The density of the connective tissue decreases slowly toward the surface. The surface is covered with a single layer of high cylindrical cells. Under the epithelium a loose network of connective tissue is seen with many plasma cells, lymphocytes and some eosinophil cells in the meshes. There is no distinct borderline between this tissue and the fibroma. The mucosa forms few low papillae and a few glandular ducts with cuboidal epithelium are seen not far from the surface. The walls of larger blood vessels are distinctly separated from the connective tissue of the tumor. In the remaining stroma of the mucosa some elastic layers are found, while in the fibroma itself no elastic tissue can be demonstrated.

Diagnosis: Diffuse fibroma; histologically benign.

Comment: Of particular interest are the history and findings of Case 2. Here we have a combination of most interesting data. *1.* The patient is a female. *2.* It occurred at the rather late age of

23 years. *3*. Pathologically a pure fibroma. *4*. Successful removal of a very large tumor with no complications or recurrence with no other means of postoperative treatment but surgical removal.

Case 1 is of interest in respect to *1*. The occurrence at the early age of 14 years. *2*. Location, attachment and size of tumor. *3*. Pathologically a pure fibroma. *4*. Its removal under local anesthesia through the nose with no postoperative bleeding or other complications.

A very marked and close relationship appears to exist between nasopharyngeal growths and infected nasal sinuses associated with chronic suppuration of the latter as seen in both cases here reported.

CONCLUSIONS.

1. The occurrence of nasopharyngeal tumors, particularly a pure fibroma, is infrequent if not rare.

2. Evidence points towards a possible relationship between nasal infections and nasopharyngeal tumors. Particularly do we consider chronic suppurative sinusitis a contributing factor in the etiology of nasopharyngeal tumors.

3. Nasopharyngeal tumors and nasal polypi may coexist, the former overlooked at time of operation, or removed together with the polypi and not recognizing its existence; mistaking the fibroma for a nasal polyp.

4. A plea is made for a better and more thorough examination before operating for nasal sinusitis associated with ethmoidal or sphenoidal polypi, in order to rule out the presence of a nasopharyngeal tumor.

5. Operative procedure to be carried out whenever possible under local anesthesia, in order to avoid operative and postoperative hemorrhages, which may occur when general anesthesia is employed.

BIBLIOGRAPHY.

1. PERSKY: THE LARYNGOSCOPE, 39:460, July, 1929.
2. COATES: *Trans. Amer. Laryngol. Soc.*, 298, 1917.
3. GOLDSMITH: *Jour. Laryngol. and Otol.*, 38:565, March, 1923.
4. HALL: *Kent. Med. Jour.*, 110, March, 1921.
5. TILLY, H.: *Proc. Royal Soc. Med.*, Dec., 1915.
6. GETCHELL: *Trans. Amer. Laryngol. Assn.*, 1909.
7. UCHERMAN: *Norsk. Mag. F. Laegevid*, Vol. 53, 9, Sept., 1916.
8. NEW and FIGI: *Amer. Otol., Rhinol. and Laryngol.*, 34:219, March, 1925.
9. MILLIGAN: *Jour. Laryngol. and Otol.*, 39:571, Oct., 1924.
10. DAHNEY: *Kent. Med. Jour.*, 20:461, July, 1922.
11. CROW and BAYLOR: *Arch. Surg.*, 6:428, March, 1923.
12. KEITH: *Kent. Med. Jour.*, 23:26, Jan., 1925.

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CHRONIC EAR DISCHARGE; DRY TREATMENT METHOD.*

Dr. E. Martin Freund, Albany, N. Y.

A frequent and at times obstinate situation facing the otologist is a chronic otitic discharge. The chronicity of the condition and the mildness or absence of other active symptoms in many of these cases are sufficiently misleading, and permit a long-standing destructive process to do irreparable damage to the involved ear. Numerous remedies and methods of treatment have been advocated and enthusiastically taken up by the profession, especially during recent years, only to be discarded or relegated to the background as ineffective procedures. Particularly has there been an absence of uniformity of results, either in improving or curing the condition.

It must be pointed out that a number of fairly effective remedies have shown promise of isolated good results. Among these can be mentioned zinc ionization, aluminum acetate ionization, mucidan irrigation, boric acid-alcohol instillation, local application of the various digestant enzymes and properly graded X-ray exposures. But none of these has given constant or uniformly good results, and at times have been quite ineffectual in clearing up chronic otitic discharges.

The underlying causes of an old but actively discharging otitis media must be approached not only from an etiological but also from a physical viewpoint. It stands to reason that the mucosa lining the external canal and middle ear becomes debilitated by the chronic irritation of a persistent aural discharge. So that aside from the causative factor, whether it be a single or multiple bacterial infection, cholesteatoma, necrosis of mastoid bone cells or even a damaged labyrinth, we must also take into consideration the recuperative power of the mucosa lining the bony and cartilaginous portions of the canal. The histological and anatomical components of that lining membrane lose their power of resistance mainly because of the destruction and granular infiltration of the delicate blood vessels supplying these parts. Thus the leukocytes cannot be called upon to play their phagocytic role at the site of infection. Another point to be considered is the large variety of bacterial flora that can be demonstrated in the

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discharge of an old otitis media. In order to overcome this multiplicity of bacteria, a powerful yet nonirritating germicide is required.

The outstanding agent that seems to meet these requirements is iodin in powdered form. Dr. M. D. Lederman has very ably and definitely demonstrated the excellent results derived from the use of iodin dusting powder (Sulzberger) in chronic ear discharges. This powder, which can be obtained from several commercial houses in this country, effectively combines the action of boric acid and iodin powder in such way as to prevent much accumulation or impaction; and furthermore, acts mainly by liberating free or nascent iodin, thus causing a nonirritating sterilization of the areas immediately adjacent to that on which the powder is applied. It is strikingly evident from the accumulated reports and from my personal experience with this substance during the past two years that the dry iodin powder has definitely proven its superior position in the treatment of chronic otitic discharges.

A reasonably large number of long-standing and very stubborn ear suppurations have readily yielded to the above form of treatment. Thus it should be the first method employed, instead of permitting the destructive process to continue over a long period of time.

A considerable part of the beneficial result depends upon the technique employed. It is of utmost importance to dry the entire external and middle ear cavities thoroughly. Unless this is properly accomplished, a good result will not be obtained. I am convinced that the lack of popularity of this method of treatment is due mainly to the ineffectual efforts to clean and dry completely the necessary surfaces before applying the powder. Total drying of the auditory canal can be accomplished in a number of ways, but best by first thoroughly wiping out the visible contents, and then by capillary suction into and beyond the perforation present. I have found the Hays suction set of considerable help in accomplishing this result. There are several other capillary suction devices on the market and one can readily perfect his technique with any of them. Good illumination is essential at all times while the suction is being applied, especially when there is a high attic perforation, or a pinpoint opening in the posterosuperior tympanic segment. Unless the perforation and the area beyond are thoroughly dried, the result will be poor and the effort wasted, for the iodin vapor is not readily liberated in the presence of a moist medium.

After the cleansing and drying have been accomplished, it is a relatively simple matter to instill the medicament by means of a powder blower that works either by mechanical air pressure or hand

propulsion. The Lederman powder blower with scoop attachment is very well adapted to this purpose. It has a number of features that recommend its use. The exact amount of the powder can be measured in the scoop and the latter can be sterilized immediately before and after use, without necessitating the boiling of the bulbous portion of the blower. I prefer the air pressure type of blower because of its long arm extension, which can be inserted deep into the canal through an ear speculum, permitting constant vision of the inner depths. The iodin powder is recommended in two strengths: a weak or approximately 1 per cent, and a strong or 2 per cent mixture.

As to the frequency of treatment required and the intervening periods, there is no definite set rule. I have found that from three to six treatments, at intervals of two or three days, usually sufficed to eliminate the most stubborn discharge. The striking benefit of this treatment becomes manifest after the second or third application of the iodin powder.

When there are aural granulations or polypi present, they must be thoroughly removed before the dry treatment is instituted. Also any coexisting conditions contributing to the chronic discharge must likewise be eliminated. Chief among these are chronic infected tonsils, obstructive adenoid hypertrophies, and dental root abscesses. Emphasis must be laid at this time on the fact that when there is definite evidence of mastoid cell destruction with a very foul discharge, surgery will have to be resorted to. In these cases, either a simple, a modified radical or a radical mastoidectomy has to be performed eventually, and under no circumstances will local treatments be of any avail.

The following is a brief description of nineteen cases which received this form of treatment under my supervision during the past two years. As will be noted, most of the aural discharges were of a duration of more than five years, while a few were rather recent, but nearly all had a discharge of a year or more.

Case 1: S. A., male, age 22 years; bilateral O.M.P.C. of over fourteen years' duration, with discharge from both ears. Small attic perforations on both drums. Progressive diminished hearing. Tonsils and adenoids removed at 12 years of age. After cleansing canals, both drums appeared pale, semifixed, edematous and partly retracted. The dry iodin treatment begun June 18, 1930. After four treatments, the discharge completely subsided and both ear canals have remained dry to date.

Case 2: B. F., female, age 35 years; discharge from right ear for past two years, with a loss of hearing on that side (28 per cent

shown with 2A audiometer). Large central perforation with granulations in margin. Right antrum and ethmoid involvement. Dry treatment commenced May 20, 1930. Local nasal attention also given. Discharge from ear ceased after two treatments and ear canal has remained perfectly dry.

Case 3: M. K., male, age 21 years; thin, yellow discharge from both ears for past few years. No pain. Associated chronic tubotympanitis with septal deviation. Ear treatment by dry method started Feb. 19, 1930. Discharge disappeared after third treatment.

Case 4: M. H., male, age 51 years; bilateral aural discharge, intermittent, for past fifteen years. Partial atresia of left canal. Marked loss of hearing and tinnitus. Left drum shows an attic perforation, while the right shows a moderate sized opening in the tubal region. Dry treatment commenced Oct. 14, 1930. No discharge after the sixth treatment. Hearing moderately improved. Tinnitus less intense and annoying. When last seen, in August, 1931, to consult about an electric hearing aid, both ear canals were perfectly dry and had been so since the last treatment.

Case 5: W. G., male, age 39 years; discharge from left ear for past fifteen years, following kick in head by horse. First seen Oct. 16, 1931, and considerable polypi seen in left ear originating in posterior-superior quadrant. Thick viscid discharge of foul odor present in left ear. Hearing loss 45 per cent on that side. There were also nasal polypi originating in left midturbinal region. Both nasal and aural polypi removed. Granulations in left drum cauterized and ear discharge continued for several weeks. Iodin powder treatment commenced Nov. 27, 1930. Complete disappearance of discharge after second treatment three days following. Patient seen April 1, 1932. Discharge had not recurred. No recurrence of aural polypi.

Case 6: F. G., female, age 5 years; discharge from right ear for past six months; gray viscid color; no foul odor; following acute otitis media preceded by acute head catarrh. No discomfort or pain in ear but discharge persistent. Iodin powder treatment commenced March 12, 1932. Complete disappearance of discharge after third treatment. Ear canal has remained clear since.

Case 7: F. H., female, age 52 years; discharge from right ear for about twelve years past, more or less constant, accompanied by scaly eczema of right external canal and auricle. Right ear drum showed high small-sized perforation with granulations around its margin. There were also chronic infected tonsils. Tonsils removed. Discharge lessened but persisted for several months. Dry iodin powder treatment instituted May 27, 1931. Ear canal dry after fourth treatment. Has remained so to date.

Case 8: C. McK., male, age 23 years; acute purulent discharge in left ear for about a week. No pain except for one day preceding onset of discharge. Left ear showed almost complete absence of drum with presence of brownish mucopurulent discharge possessing no foul odor. After several iodin powder treatments, begun Dec. 31, 1931, discharge disappeared. The drum commenced healing. When last seen, Jan. 27, 1932, the ear canal had remained dry and hearing returned to normal from a previous loss of hearing of approximately 18 per cent.

Case 9: V. W. J., female, age 43 years; moderate discharge from right ear for one and one-half years following series of head colds and right-sided otitis media. Discharge had been constant though not copious, no foul odor, worse during winter months, not accompanied by pain or discomfort. Small perforation seen in midportion of right drum with some granulations in margin. Drum moderately thickened. Treatment commenced Jan. 8, 1932. Two treatments sufficed to clear up discharge and when last seen for check-up, May 1, 1932, right ear canal completely dry.

Case 10: D. G., male, age 3 years; bilateral aural discharge since 2 years of age, following spontaneous rupture of both drums, with glandular swellings, both sides of neck. No mastoid development. Nasal discharge also present, though not profuse. Discharge from ears mucopurulent with excoriation of lining of canals and skin of both ear lobes. Treatment commenced May 30, 1930; three treatments being sufficient to clear up the discharge. The nose and throat were also treated. Ear discharge had not recurred.

Case 11: M. L., female, age 7 years; showed a chronic otitis media of right ear, duration three months, following spontaneous perforation at site of old previous myringotomy. Large central perforation seen in right drum with yellow mucoid discharge under pulsation pressure. No foul odor. Tonsils and adenoids had been removed several years before. One iodin powder treatment, given July 18, 1931, brought about disappearance of discharge. Child seen one month later; showed right ear canal perfectly dry with perforation commencing to granulate.

Case 12: G. R., male, age 41 years; discharge from right ear for past four years, intermittent in character, worse when patient was tired or had overworked. Several small perforations seen in right drum, one in attic region. Had various treatments on numerous occasions since onset of discharge, but without success. Chronic hypertrophic tonsillitis noted; tonsil removal advised, which was done under local anesthesia. Iodin powder treatment commenced one

month later, Feb. 24, 1931. After four treatments discharge completely disappeared and had not returned when last seen for check-up, May, 1932.

Seven more cases of this series gave similar excellent results, making a total of nineteen cases of chronic ear discharge which have been observed within a period of two years. These cases, as indicated, had a more or less continuous discharge from either one or both ears, ranging over periods of several months to 12 or 15 years.

Recently interest has been renewed in the zinc ionization method. But the dry method herein described is simpler, and certainly more productive of permanent results.

BIBLIOGRAPHY.

ASHERSON, N.:: THE LARYNGOSCOPE, Feb., 1932.

BECK, J. C.: *Ann. of Otology*, Dec., 1929.

FOTIADA: *Monatsschr. f. Ohrenheilk.*, 1926.

GUTTMAN, J.: *Illinois Med. Jour.*, Aug., 1927.

LEDERMAN, M. D.: THE LARYNGOSCOPE, Nov., 1917.

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IODINE INJECTION THERAPY IN OTO-RHINOLOGY. A NEW METHOD OF TREATING NASAL AND AURAL CATARRHAL CONDITIONS.

Dr. Joseph Rosenberg, New York.

The obscure etiological background of acute and chronic catarrhal conditions of the nose and ear obviously restricts us to their consideration within the confines of manifestations and symptomatic treatment. Protracted forms of coryza, vasomotor rhinitis and otitis media catarrhalis acuta form a very important pathologic group with which every otolaryngologist is frequently confronted. Moreover, these conditions will at times prove exceedingly unyielding to the most drastic therapeutic measures. Any progress that is being made in respect to these measures which will materially aid us in coping with this problem is therefore deserving of more than passing consideration. I propose to present herewith a method of treating these catarrhal conditions which thorough clinical tests have demonstrated to be distinctly superior to the commonly employed treatments.

Symptomatology: Acute catarrhal rhinitis, usually termed coryza, is one of the most common ailments of humanity. It manifests itself subjectively by spasmodic sneezing and considerable watery discharge caused by the excessive accumulation of fluid in the membrane. Rhinoscopic examination will reveal a turgescent mucosa, which is not, however, inflamed or otherwise discolored in the early stage. Respiration is impeded, due to the occlusion of the nasal passages, and frequently headache is an associated condition.

If the vasomotoric dysfunction is allowed to continue unabated, the catarrhal symptoms may gradually be superseded by an infectious condition marked by inflamed membranes and copious purulent discharge. This condition, which may be brought about by the lowered resistance of the nasal mucosa, permitting the invasion of pathogenic saprophytes, is beyond the scope of this paper. It is of interest, however, insofar as it, at times, may lead to a subacute form of coryza marked by tumefaction and continued hypersecretion from the nasal mucosa. This protracted form of coryza may last for weeks or even months, and is not to be confused with vasomotor rhinitis or hydrorrhea, due to pyogenia bacteria.

In vasomotor rhinitis, an objective examination may prove anatomically negative. The nasal mucosa will, however, often assume

a bluish-gray discoloration. Subjective manifestations, such as sneezing and copious watery discharge, are chronic in certain cases, whereas in others they are apt to recur spasmodically (with radical temperature changes, exposure to draft, etc.). The highly allergic forms of vasomotor rhinitis, involving asthma, are notably refractory and constitute one of the most perplexing problems of immunology.

I now employ the same therapeutic and palliative measures in the treatment of catarrhal rhinitis, hydrorrhea and vasomotor rhinitis. This is a natural procedure to follow when it is borne in mind that what we primarily endeavor to attain is symptomatic relief. That the prognosis may differ in cases of vasomotor rhinitis of years standing is granted; but I claim that any method of treatment to which this very refractory condition may at times yield is fully justified as a nonspecific measure.

Likewise, in otitis media catarrhalis acuta and cases where the drum membrane is perforated, a thin watery discharge is frequently present. This condition is apt to be a sequel to acute catarrhal attacks of the nasopharynx and is marked by hyperemia and turgescence of the mucous membrane in the nasal cavity, increased secretion and exudate. Objective examination will also reveal redness. In the milder cases the mucosa will, as a rule, return to its normal condition, whereas in severe or neglected forms, chronic abnormalities in the tympanum, such as tubal stenosis, may be the result. Subjectively, the patient will complain of impaired hearing and buzzing in the head.

Treatment: So much has been said and written about iodine and its therapeutic properties since its discovery as a substance by Courtois, in 1811, that any historic review is apt to become tedious repetition. The palliative and therapeutic value of oral administration of tincture of iodine and potassium iodide (KI), as well as intravenous injection of sodium iodide, are familiar remedies of many years' standing. As a matter of fact, iodine therapy has hardly made any noteworthy progress in several decades.

To Sternberg belongs largely the credit for developing in recent years a new form of iodine therapy which, in my opinion, constitutes a valuable contribution worthy of the consideration of every otolaryngologist.

Sternberg demonstrated that while the old methods of iodine application, though quite irritating, might be of value at the very onset of the coryza, they proved entirely ineffective in the subsequent pathogenesis. On the other hand, it appeared that subcutaneous injections of small amounts of sodium iodide containing traces of free iodine would elicit remedial qualities hitherto unknown. I have ad-

ministered these injections in the most refractory cases of coryza and catarrhal conditions of the middle ear with rather astounding benefit.

There is now available in this country a preparation identical to that used by Sternberg. It is called iodomin. Each ampoule of iodomin contains a 1 c.c. solution of 5 per cent sodium iodide with traces of free iodine. The pure iodine content is exceedingly low, precluding the possibility of iodism or untoward reactions, except for a burning sensation at the point of injection, which lasts for about five minutes. When conducting a test for the specific purpose of verifying claims made for a new method of treatment, we are always prone to select the most obstinate cases where the usual measures have proven unsatisfactory or would be likely to disappoint. Most of the patients whom I chose for the purpose of testing iodomin belong in this category.

I had, first, occasion to administer iodomin in a series of middle ear conditions, all revealing the characteristic catarrhal symptoms. They were refractory types of several months' standing. Subcutaneous injections of iodomin were administered two or three times a week. Some cases responded quickly, after the second or third treatment, whereas others would be less yielding, requiring as many as eight or ten injections. The results as a whole were highly gratifying.

My subsequent experience with iodomin in a number of cases of coryza and nonspecific rhinitis was equally satisfactory. Needless to say, these were all conditions which had failed to yield to the customary remedial measures. Only rarely is it our good fortune to be assigned a case of acute catarrhal rhinitis at its onset. Not until the condition has assumed seemingly chronic characteristics will the great majority of patients seek professional aid. Thus it will appear natural that every case of coryza included in my iodomin test was of several months' standing, although none of them was sufficiently conspicuous to justify a detailed report.

I have never found it necessary to administer more than eight or ten injections of iodomin. Gradual or abrupt cessation of the subjective symptoms is the first manifestation of improvement elicited by its exhibit. Occasionally the patient is relieved of sneezing spells within a few hours after the first treatment has been administered. A gradual diminishing of secretion from the mucosa and abatement of the pathologic symptoms then follow. Coryza, even in its most protracted form, is likely to yield more readily to iodomin treatments than is hydrorrhea. Whereas the former is apt to respond after the

second or third injection, vasomotor rhinitis, even of the mildest type, will require from six to ten treatments. I wish to stress the fact here that I have not as yet had occasion to demonstrate the therapeutic merits of iodomin in the severest types of vasomotor rhinitis, although I have every reason to believe that these injections will meet the requirements of an effective nonspecific remedy.

CONCLUSIONS.

1. A brief review is presented of the objective and subjective symptomatology of catarrhal conditions of the nose and ear.

2. Little noteworthy progress has been made in several decades in respect to the treatment of these common ailments, which frequently are found to be highly refractory.

3. In my experience I have found that iodine injection therapy constitutes a decidedly improved method of treating catarrhal conditions of the nose and ear.

4. A series of cases of protracted coryza, vasomotor rhinitis and otitis media catarrhalis acuta were treated with iodomin—a subcutaneous injection of 5 per cent sodium iodide containing traces of free iodine.

5. Iodomin injections will produce relief of subjective symptoms, followed by a more gradual objective response. Sometimes a few treatments will suffice in the milder cases of coryza and otitis media catarrhalis acuta, as many as eight or ten treatments may be required in the more refractory types and vasomotor rhinitis.

6. The therapeutic value of iodomin is predicated upon the presence of a characteristic catarrhal condition, and is therefore worthless in the treatment of infectious colds of pyogenic origin or wherever an anatomical deficiency is the underlying pathologic cause.

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LARYNGEAL STENOSIS — GROWTH OF THE LARYNX AS A FACTOR IN TREATMENT.

DR. CHEVALIER JACKSON and DR. CHEVALIER L. JACKSON,
Philadelphia.

A review of the literature on the treatment of stenosis of the larynx seems to indicate the overlooking of what is one of the most important factors in the treatment of stenosis of the larynx in children, namely, the growth of the larynx. Another thing that does not seem to be realized is that because of this factor of growth, laryngeal stenosis in children is in an entirely different class of case from laryngeal stenosis in adults.

In adults surgical methods must be planned upon conservation of what cartilage may remain unimpaired by the primary disease or by the previous, ill-advised surgical efforts to cure the stenosis. In children it is not only necessary to conserve all cartilage but also to take advantage of the opportunity to force the growth of the immature larynx; and, above all, *to observe the utmost conservatism as to all surgical measures in dealing with the larynx of children.*

The fundamental rule in children should be: *Maintain a lumen and force the child to breathe through it.*

The forcing of the child to breathe through the larynx is accomplished by graduated, continuous corking of the cannula.[1, 2, 3]

It is not intended here to go into details as to methods of treatment; they have been elsewhere considered;[1, 2, 3, 4] but rather to stress the overlooked factor of forcing the growth. Supplementary to this, the next most important point, namely, conservatism in the removal or destruction of any tissue found in the stenosed larynx of children, is also often overlooked. Increase in size of the lumen of the larynx that comes with growth aided by direct laryngoscopic dilatation, or, in the more difficult cases by the elastic pressure of increasing sizes of smooth rubber tubing (see Fig. 1) will in time usually take care of any cicatricial or redundant endolaryngeal tissues (see Fig. 2).

It is distressing to note that insistence upon the great aid obtained in the treatment of laryngeal stenosis in children by the growth of the larynx has been so misconstrued as to merge into the advice so

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often ill-advisedly and casually given in various diseases of childhood, namely, "He will outgrow it." It cannot be too emphatically stated that *children never outgrow stenosis of the larynx*. Increase of lumen in the stenosed larynx of a child occurs along with growth only when the child is forced to breathe through the larynx. In the treatment of laryngeal stenosis this can be obtained only when a lumen sufficient to permit wearing of, at least, a partial cork in the tracheotomic cannula has been obtained. To allow a child to drift along wearing an uncorked cannula by telling the parent "he will outgrow it" is to lose the best possible time for cure. As previously pointed out, for proper, proportionate growth of the larynx it is

Fig. 1. Closed-end rubber laryngostomy tube of Chevalier Lawrence Jackson for the treatment of laryngeal stenosis. The tubing is glass-moulded and has a specially shaped upper end that fits under the epiglottis so as not to cause any irritation or discomfort at the upper orifice of the larynx or in the laryngopharynx. The closed end prevents food or liquids getting into the airway.

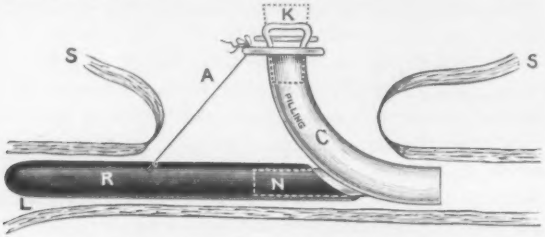

Fig 2. Rubber tubing with closed end for treatment of stenosis of the larynx in children. The tubing is here shown attached to the standard laryngostomy apparatus;[1] but the tubing is made in various forms adaptable to all methods of treatment of laryngeal stenosis. The OPEN end is cut off to make the tubing of the exact length required for the particular case. The tubing is obtainable in any diameter required. The use of slightly increased sizes in succession at suitable intervals is fundamental for best results.

necessary that aerial respiratory excursions in and out be continuously maintained. If the strictured lumen is small a sufficient amount of air cannot be obtained through the larynx. In such cases a partial cork must be worn in the cannula and the by-passage for air must be ground on the side of the cork of proper size to admit just the additional amount of air necessary and no more.[1, 2, 3] If this by-passage is gauged with proper nicety the larynx will grow in proportion to general growth in the same way as the larynx of the normal child. If, on the other hand, an abundance of air is admitted through the neck the larynx will remain undersized; though it may not remain stationary in general dimensions, the lumen will not increase

in the relative proportion to growth that would be the case in the normal individual. In some cases the atresic larynx may even, for a long time, remain infantile in size from disuse. In gauging the size of the by-passage, though slight indrawing at the epigastrium is desirable, the by-passage must not be so small as to create a funnel-breast, either temporarily or as a fixed deformity.

CONCLUSIONS.

1. The greatest single factor in the successful treatment of stenosis of the larynx in children is the increase in size of the larynx during the period of general growth of the child.

2. To obtain the utmost advantage of growth, however, it is necessary to maintain a lumen and to force the child to breathe through it, by partial corking of the cannula. The larynx will not grow properly or adequately if the child is allowed to breathe freely through the neck.

3. In most cases the lumen can be maintained by direct laryngoscopic dilatation, combined with graduated continuous corking, over a long period of time. In a few cases other methods by rubber tube or otherwise are necessary; but whatever the method, the most important thing is to force the growth of the larynx.

4. The utmost conservatism as to surgical procedure is fundamental in dealing with the larynx of a child. Radical measures, especially when accompanied by the removal of tissue or destruction of cartilage by secondary suppuration, may ruin the larynx beyond hope of cure.

BIBLIOGRAPHY.

1. JACKSON, CHEVALIER: Orthopedic Treatment by Corking the Tracheotomic Cannula. *Jour. Laryngol., Otol. and Rhinol.*, London, Vol. XXXII, Feb., 1917.

2. JACKSON, CHEVALIER: Peroral Endoscopy and Laryngeal Surgery. Textbook. THE LARYNGOSCOPE, St. Louis, 1914.

3. JACKSON, CHEVALIER: Bronchoscopy and Esophagoscopy, 2nd Ed., W. B. Saunders Co., Philadelphia, 1927.

4. JACKSON, CHEVALIER, and JACKSON, CHEVALIER LAWRENCE: Surgery of the Larynx, Trachea and Bronchi. Dean Lewis' Practice of Surgery, Vol. IV, Chap. 6, pp. 1-232.

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AMERICAN OTOLOGICAL SOCIETY.

(Continued from October issue.)

DISCUSSION.

DR. ARTHUR B. DUEL: Dr. Kerrison's paper is a strong plea for more research into the problem of meningitis. He advocates the method of Louis Weed, who has discovered a simple method of reducing intracranial pressure. Kubie's careful experiments on animals, pointing to the value of intrameningeal drainage, may be shown to have great clinical importance. Meningitis is not a peculiar disease, belonging only to the meninges, but is one that is applicable to the same broad surgical principles observed in other parts of the body. An additional difficulty, however, is that the meninges are inclosed in a bony capsule. Methods of craniectomy at the present time are not greatly in advance of neolithic surgery. It is urgent, therefore, that craniectomy should be made the subject of careful experimentation and improvement in technique.

We must bear in mind that meningeal inflammation may be local or diffuse; intradural without involving the arachnoid spaces, or involving both. These varying types present differing degrees of menace to the patient. The simpler forms, which if operated in time, are within our power to check, may if unoperated, progress into the graver forms beyond our power to check.

To summarize, we must emphasize the following points: 1. Early diagnosis; 2. immediate operation; 3. adequate drainage; 4. renewed effort at improved technique.

DR. LAWRENCE S. KUBIE: In regard to the question of so-called "forced drainage" the following points of meningeal drainage should be borne in mind:

1. Necessity of early drainage, before walling off occurs in the intracranial spaces.

2. Necessity, not only of surface drainage, but of the depths of the central nervous system as well.

3. Opening of the skull may cause collapse of the meninges, or a mass settling of the whole brain on the base, causing obstruction to outflow of fluid.

4. Necessity of prevention of this collapse by forcing new production of cerebrospinal fluid.

5. Normally, cerebral pressure is maintained by balance of fluid transuded through the capillaries and that reabsorbed by the venous capillaries. If, however, intracranial pressure is reduced to atmospheric pressure by drainage, and at the same time production of cerebrospinal fluid is stimulated by lowering osmotic pressure of the blood, we can get fluid movements from the depths of the brain to the surface and out through the operative drainage tract.

6. There are various methods of drainage, none so far perfect, but the most important seems to be lumbar drainage with the head high, so as to drain the whole tract.

7. Drainage alone cannot effect a cure but must be supplemented by adequate surgical procedures, and promotion of fluid.

8. Promotion of fluid is best effected by intravenous injection of hypotonic solution. The injection is made slowly to prevent overloading of the circulation with strain on the heart.

9. Difficulties in the way of drainage are: a. the thick gelatinous exudate which forms in the meninges; b. the formation of pockets of infection. The first difficulty will have to be solved by proper dilution of the exudate. The second point will depend on individual procedures. Recently, Dr. Leon Cornwall, of New York, used air injection to break up obstinate adhesions, with the result of successful evacuation of a large quantity of pocketed pus.

10. In conclusion, we may consider the nasal cavity as a means of possible approach to the base of the brain. This is a pathway by which a very continuous system of forced drainage might be established under the proper technique.

DR. PHILIP D. KERRISON (closing): Dr. Kubie's discussion was particularly interesting for the reason that what he says with regard to forced drainage may be regarded as authoritative. Dr. Duel in his discussion referred to the surgical analogy between the treatment of otitic meningitis and that of suppurative processes in other parts of the body. In theory, at least, that is a point of considerable importance; for if we run over mentally all the known lesions due to infection, we shall find it difficult or impossible to recall a single suppurative lesion, unless it be that of a suppurative endocarditis which is not in some degree amenable to surgical treatment. Is suppurative leptomeningitis the one other exception, the one other suppurative lesion which will forever remain beyond the scope of surgical therapy? Combining logical surgical methods with the forced drainage theory of Kubie, I personally believe that the reverse will prove to be true.

Dr. Eagleton speaks of different types of meningitis, some of which he says, dogmatically, are hopeless. I would call Dr. Eagleton's attention to the fact that at the present time experiments and investigations in this field are being carried on by several highly scientific groups, and that it is highly unscientific to state in advance what the ultimate results of these experiments will be.

With regard to carotid ligation, Dr. Eagleton brings to the support of the procedure simply his unsupported work and belief that it is a wise therapeutic measure. He thinks that he has beneficial results from it in cases of otitic meningitis. Some of us hold exactly the opposite opinion.

Naturally the first and most obvious argument against it might be the fact that it violates the generally accepted physiological law that an unimpeded blood supply to a diseased part is one of Nature's chief instrumentalities for bringing about resolution and recovery. I prefer, however, to base my argument against it on somewhat different grounds.

Both Dr. Eagleton and I are interested in the treatment of meningitis. But neither he nor I, I take it, would claim to be an authority on the subject. No man is. Neither he nor I, therefore, can expect a purely personal opinion or even a personal statement of experience to carry much weight or to survive, if it is in opposition to the consensus of opinion held by scientific workers and physiologists generally. Now, modern scientific thought is overwhelmingly behind the thesis, or proposition, that the cerebrospinal movement is in itself the instrumentality through which, if ever, purulent meningitis will be brought under therapeutic control.

In one of Dr. Eagleton's papers he discussed at some length the so-called "cerebrospinal circulation," and the agencies through which it is initiated and maintained. His conclusion, which of course is the obvious one, was that next to the hydraulic pressure of continuous production, the cerebral fluid movements are maintained chiefly through the bulk movements of the brain itself, which brain movements are activated chiefly and probably solely by arterial pulsations. When, therefore, he proposes as a therapeutic measure in purulent meningitis to ligate the common carotid he suggests a proceduring which should tend: 1. to diminish the production of fluid in the ventricles and in the perineural spaces of the central nervous system, 2. by reducing arterial pulsations to inhibit or reduce the brain movements by which the cerebral fluid is kept moving, and 3. as a byproduct of the mechanical result of No. 1 and No. 2 to promote cerebrospinal stasis in the germ laden cisterns at the base of the brain, and in all the perineural spaces throughout the central nervous system. Viewed in this light, carotid ligation seems not only to be sadly illogical, but a violation of modern scientific thought and theory, which is, to say the least, somewhat archaic.

Dr. Eagleton states that a prerequisite to successful treatment is the determination of the pathology by which the infection has taken place. It would of course be highly desirable if this could always be done. But we have each and all of us, in the course of our otological experience, seen and studied many cases of otitic meningitis; and I think the members of this Society will bear me out in the statement that the cases in which the pathway of infection can be definitely determined are the exceptions rather than the rule. Dr. Eagleton is, therefore, insisting upon what we all know to be the vast majority of cases, an impossibility.

Experimental Investigation of the Physiology of the Ear, Using the Method of Wever and Bray. S. J. Crowe and Walter Highson.

At the meeting of the American Otological Society in June, 1931, the authors presented the results of their first year's work on the normal and pathological physiology of the ear as studied by the method of Wever and Bray. During the first year only the simplest equipment was used and the conclusions drawn were largely subjective impressions. At this meeting the equipment used for the more accurate recording of experimental results is described and charts are shown of the relative intensity of the transmission of speech and of pure tones (from 250 to 4,000 cycles) before and after the production of experimental lesions of the various structures in the middle ear. Both air and bone conduction have been studied with this method.

The apparatus is described in detail by E. G. Witting in an article now in press. A general description of the first year's work was published in the *Zeitschrift fur Hals-, Nasen-, und Ohrenheilkunde*, Vol. 30, Heft I, 1931, and in the *Proceedings of the Interstate Post-Graduate Medical Association of North America*, Vol. 7, 1931. The plan of investigation of the Otological Research Laboratory and some of the more important physiological conclusions from the study of the human material and the animal experiments were presented before the Harvey Society in New York in January, 1932. The experiments on the effects of immobilization of the round window membrane were presented by Walter Hughson before the American Association of Anatomists in March, 1932, and will be published in the *Annals of Otology, Rhinology and Laryngology*.

DISCUSSION.

Dr. E. G. WEVER: I wish to speak briefly on a point of method, in regard to determinations of intensity, which, though closely comparable to that of Dr. Crowe, has a difference in the switching arrangement, which we consider advantageous; and by which one observes alternately the responses from the nerve and the standard sound, the arrangement being such that when the standard sound is equal in loudness to the sound from the animal, one hears a continuous sound instead of a varying one as the switching is made. The two sounds are observed against the same background of noise constantly present, and quantitative determinations may be made with considerable precision.

MR. R. L. WEGEL: The questions raised by Dr. Crowe's work are principally questions of mechanics. He is studying the performance of the middle ear, using the nervous tissue more or less as he might use a microphone to detect vibrations. Having thus largely limited himself to a study of the middle ear, his results bear on more specific questions, and insofar as he has actually eliminated the nervous tissue, his results can be discussed in terms of well known principles of a quantitatively unknown problem.

Most of the observations which he has reported are in accord with what is to be expected on the basis of a crude knowledge of the mechanism, but there are certain results which seem to contradict this simple type of reasoning. This is by no means surprising as anyone who has had experience in the quantitative design of acoustical devices will know. From a rigorous standpoint the middle ear mechanism may be thought of as constituting six possible channels for transmission of energy from the ear to the labyrinth, each channel corresponding to some combination of the six possible motions of each one of the elements of the chain. The ossicles are so attached that these six ways of transmitting energy are not independent. This mechanism is roughly analogous with six telephone lines, all connected to a transmitter at one end and a receiver at the other end. These transmission channels, in order to carry out the analogy, must be close enough together so that there is a considerable amount of "cross-talk" between them. When Dr. Crowe applies a pledget of cotton to the round window he must, rigorously speaking, effect all six of these channels and thus, while it seems that he is providing one mechanical constraint, he is actually applying a minimum of six constraints. If the quantitative constants of the middle ear were accurately known, they would no doubt show that of the six channels, only one, or perhaps two, are of any practical

importance in transmission of sound except possibly in certain types of pathologic condition. Each of these channels is of the general type of band pass filter which transmits sound within a certain frequency range more efficiently than others and the combination of the six channels is also a "band pass" structure. This follows from a mere inspection of the parts and is in accord with the observed fact that the ear is most sensitive within a frequency range 1,000 to perhaps 5,000 cycles. Whether or not a working concept of the mechanism is most advantageously stated in such terms as I have given or not, the fact remains that in order to find out what to expect when a given lesion exists, it will be necessary to solve the mechanics problem as I have stated it. When this is done there will be no unexpected results obtained by mechanical blocking of certain parts. Dr. Crowe frequently speaks of the action of the pledget of cotton on the round window as blocking its motion. This is an unfortunate term to use for it suggests that the round window is thereby prevented absolutely from moving. It will be fairly obvious that the addition of a pledget of cotton cannot have an effect greatly different in order of magnitude than that of the cochlea itself. Dr. Crowe's results of course have, as I understand him to say, a direct outlet in the treatment of hearing defects, quite regardless of any theory, but those of us who are primarily interested in finding the mechanism will be much interested in following Dr. Crowe's continued work along this line.

DR. D. HAROLD WALKER: For years, through the suggestion of Dr. Clarence Blake, I have been placing cotton pledgets in the niche of the round window for the purpose of improving hearing. The hearing apparently is improved by the blocking of the round window and by not allowing any vibrations which go to the organ of Corti by means of the stapes to escape.

DR. EUGENE R. LEWIS: I do not see how hearing was improved by blocking the round window. Did I understand that correctly?

DR. CROWE: Yes, the hearing was improved.

DR. EDMUND P. FOWLER: Did Dr. Crowe notice any difference in the quality of the sounds after blocking the round window? If this was due to reflection of sound, there would be some slight alteration of the tone quality.

DR. CROWE: It was much clearer and more distinct. The increase was from three to 15 feet.

There is a very simple experiment that demonstrates the *improvement* in bone conduction. In the cat, the bulla and middle ear is opened through an incision in the neck; air conduction and bone conduction are tested and charted as described in previous publications. The middle ear and bulla is then filled with mercury. This fixes both the stapes and round window membrane. Under these conditions air conduction is nil, but bone conduction is actually and measurably improved.

Electrical Phenomena in the Auditory Mechanism. Dr. L. J. Saul, Dr. H. Davis and Dr. M. H. Lurie.

A method employing modern neurophysiological technique is used for the study of electric acoustic phenomena. Two effects are distinguished: true nerve action currents and a spreading electrical effect from the cochlea.

The true action currents have been traced along the auditory tracts from the VIIIth nerve to the auditory radiations. They are sharply localized, follow contralateral tracts, are reversibly deleted by anesthetics, and disappear immediately on sectioning the VIIIth nerve or at death. Using musical tones as stimuli the pitch applied to the ear is recovered only up to about 1,000 double vibrations per second. Words are not intelligible; probably because the higher pitches are lost.

The "spread," in contrast to the action currents, is a diffuse electrical broadcast which probably originates in the cochlea and which spreads through all the tissues of the head. It is probably in no way concerned with physiological transmission from ear to brain. It shows a point of maximal response in the posterior groove of the subarcuate fossa. It is much more resistant to anesthetics than are action currents. It may persist for several minutes after death. In one experiment it gradually faded out over a period of 20 minutes after completely cutting the VIIIth nerve and cochlear artery. In response to musi-

cal notes applied to the ear, tones may be recovered up to at least 4,000 vibrations per second. The lower limit, at a given intensity, is not so low as for action currents. Words are reproduced very clearly.

Some preliminary technical observations are presented. The action current appears in the VIIIth nerve, about five to eight ten-thousandths of a second after the peak of the sound wave. Stimulation seems to occur at the peak of the sound wave. The intensity threshold for action currents appears to be lower than for spread. Their relation to intensity of stimulating tones seems to be logarithmic while that of spread is linear. Strong harmonics are evident in the spread wave at certain frequencies.

A cat exposed to a tone of 600 d.v. for three months, and another for six months, showed no tone islands. The latter cat showed a definite maximum at 600 d.v. in the action currents from brachium and auditory radiations, but no maximum from VIIIth nerve or spread.

We conclude tentatively that the upper limit of frequency of action currents in a nerve fibre is probably not over 1,000 per second.

DISCUSSION.

DR. LORENTE DE NO: Dr. Saul and Dr. Lurie have determined the frequency of the potential changes in different parts of the acoustic tract. I would like to know if the frequency of their "true action currents" is the same in the VIIIth nerve as in the medulla and in the midbrain, or if there is an indication of change of frequency when the impulses pass through one of the nuclei of the acoustic tract (the ganglion ventrale and tuberculum acusticum, the oliva superior complex, the posterior colliculus, the corpus feniculatus internus). If the electrical changes recorded by Dr. Saul are true action currents, it is most likely that they change their frequency when the impulses pass through the different nuclei. In the cochlea we have several different kinds of endings, of which the most important are the fibres for the internal hair cells, of which it may be said that there exists a fibre for each hair cell, or for a few closely lying cells; and the spiral fibres that are connected with very numerous external hair cells. Any vibration of the membrana basilaris will set up impulses in many fibres and, of course, not simultaneously. A few fibres that each conduct a small number of impulses per second can give the picture of impulses traveling at a very high frequency if the fibres are not synchronized, as most likely happens in the cochlear nerve.

DR. D. HAROLD WALKER: If the physiologist can tell us how the sound is taken from the organ of Corti to the cerebrum, and if the physicist can devise a method by which sound can be transmitted to the organ of Corti by some other method than through the middle ear, then the problem of deafness due to adhesive changes in the middle ear might be solved.

PROF. TAIT: I notice Dr. Saul says that the electric charge reached the brain and spread all over, but the action currents proper were quite localized. He traced them up to their final termination in the cortex. Is the rhythm retained in spite of the interposition of other nuclei on the pathway? Dr. Saul says he could put a needle in subcortically and could pick up action currents at their termination. His exposition concerned particularly the preliminary nerve-conduction stage, rather than the final cortical stage.

DR. E. G. WEVER: In regard to the stimulation-deafness problem mentioned by Dr. Saul, I can add two more negative cases. We stimulated two cats with very loud sounds for three weeks, but found on testing them no evidence of modification of sensitivity. We suspect on the basis of previous studies in this field that certain animals are more susceptible to the destructive action of sounds than others. Horton, working in our laboratory, is at present developing the conditioned response method with guinea pigs to a point where audiometric curves can be taken. In seven animals, thus far tested, he finds the hearing is very much like that of man, with no greater differences than would be apparent in seven human subjects selected at random.

DR. MAX A. GOLDSTEIN: If the physiologist places the maximal rate of vibrations at 1,000 when he measures nerve action currents, how can we hear 30,000 d.v.? If he measures 1,000 d.v. or less through any nerve impulse, what right has he to presume that any other impulse may be measured in any other way?

DR. J. LEON SAUL (closing): Our observations support Dr. Crowe's, and we believe he is working primarily with spread, which, in the vast majority of cases, is an excellent index of what the animal is hearing. In answer to Dr. de No, we get the same frequencies up to the acoustic radiations. As to what happens in the cochlea when sound is transformed into electrical impulses, this is only hypothetical, and more than one mechanism may be found to fit the facts. Dr. Goldstein's question plunges us into the problem of auditory theory. We can outline possibilities, but cannot say what actually happens.

Researches on Labyrinth Reflexes Dr. Rafael de No.

Dr. de No gave a summary of the results of his study of the anatomy and physiology of the labyrinth and vestibular apparatus. In the first part he described the most important characteristics of the vestibular reflexes, recorded by the eye muscles of the rabbit (postural reflexes, rotation reflexes and reflexes due to excitation of the individual semicircular canals).

In the second part is described the anatomy of the peripheral and central endings of the vestibular nerve.

In the third part he mentioned some experiments on the physiology of the vestibular pathways in the medulla and midbrain and presented two diagrams which may explain two of the most important features of the vestibular reflexes: the after-discharge and the appearance of the quick phase of the nystagmus.

DISCUSSION.

PROF. TAIT: Dr. de No has been working at the equilibrial part of the labyrinth along different lines, but on lines that give results similar to those obtained by Dr. McNally and myself. Dr. McNally has cut nerves without interfering with the membranous labyrinth at all, so that we can have a frog in which one, two, three, in fact any number of canals are lacking. He can leave a single canal or any combination of canals intact. Thus a negative type of experiment in the one case can be checked by its corresponding positive in the other. We have found that the whole apparatus of the labyrinth, the six canals and the two auricular maculae, work as one unit in maintaining and controlling equilibrium.

DR. J. GORDON WILSON: Dr. de No's paper is a physiological investigation of the labyrinthine role in compensation. During movements of the head in space the eyes of normal individuals make adequate compensatory movements necessary to ensure precise vision. In the eye muscles action necessary to give this in spite of bodily movements, the labyrinth plays a part.

The fixation of the eye for vision is a very perfect mechanism and in all voluntary movements all six eye muscles in each eye take a part as agonist, antagonist and fixation muscles. Also in movements of the eyes from labyrinthine stimulation all the muscles participate to give the appropriate deviation.

When we turn attention to the labyrinth and enquire what is happening, we find ourselves debarred from direct observation. Maxwell, in the dogfish, could by direct observations on the essential parts of the labyrinth see what he was doing. In the frogs and birds, Tait and McNally are coming close to this. But in mammals we are stimulating a peripheral organ not so directly available and our physiological conception of what occurs is influenced more or less by a deduction from studies on lower vertebrates based on mammalian anatomical knowledge.

While it is agreed that there are two muscle reactions from labyrinth irritation, *a*. kinetic or dynamic; *b*. static or postural, the anatomical position of these reactions is disputed as well as the physiological connections involved. Maxwell showed in the dogfish the ampullae alone suffice for all the dynamic and all the static postural functions of equilibrium originating in the ear and that otolith organs of the utricle suffice for all static and dynamic functions of equilibrium except response in a horizontal plane. Magnus believed that the dynamic reaction originated in the ampulla of the canal and that the excitation of the maculae of the utricle and saccule gives a tonus or static effect. Tait and McNally believed that the semicircular canals are not involved in static reactions, they respond only to acceleration and only give compensatory move-

ments; the utricle is the apparatus for static equilibrium; de No's experiments tend to support Maxwell.

To me the most interesting part of the paper has been the neurological. The diagrams of the nerve endings are interesting and convincing. Dr. de No's diagram to explain why the nystagmus outlasts the labyrinth excitation (which, it may be remarked, is but one example of a phenomenon so characteristic of reflex contractions) appears to me as satisfactory as any yet offered and well in keeping with the results of Ranson's experiments.

PROF. PARKER: Trophic transmission over nerve fibres probably takes place by the passage of substance along the nerve. Such substances may escape at the nerve terminals, and in this way the nerves may become secretory. Probably this function permeates the whole nervous system, the secretory activity taking place at the synapses. Chromatophores respond to such secretory material long after the true nervous action has ceased. The retardation mentioned by Dr. de No may thus possibly be approached.

DR. DE NO (closing): If that is true, that would settle the discussion. There would be some mechanism to take away or compensate for the secretion. We need both substances to complete the explanation.

We must, of course, be very careful in translating the results of experiments on rabbits to the human physiology. The eyes of the rabbit show strong tonic reflexes that in man are almost missing and, on the other hand, the rabbit does not have optical reflexes of any importance. Then we must consider that the labyrinth is prepared to produce normal reactions only when it is excited under normal conditions. The nystagmus compensates the rotation of the head only when the rotation is a physiological one; *i. e.*, when the rotation corresponds to any of those that are used by the rabbit itself. After-nystagmus is never physiological. In my papers I have described a "physiological rotation" that sets up rotation nystagmus, but not after-nystagmus.

The diagrams attempting to explain the production of after-discharge and of the quick phase of the nystagmus have been developed in many directions; they are perfectly consistent with each other if we consider that the neuron chains may have quite different thresholds, and therefore may need different times for setting up a discharge. The experiments of Dr. Tait and Dr. McNally will certainly be of greatest importance for the physiology of the labyrinth. I am glad to have heard their results in advance.

I agree with Dr. Parker that chemical changes must be the primary cause of most of the nervous phenomena. The theory of an exciting and inhibitory substance producing antagonistic effects would explain most of the results of the experiments that I have carried out.

The Use of Various Types of Audiometers in Clinical Work. Dr. C. C. Bunch.

Clinically, hearing tests are designed to assist in diagnosis and to help determine the effects on hearing of any treatment which may be instituted. A clinic in which teaching is a primary function must also take into consideration the training of those being taught (internes, assistants, etc.). More than this, the correction of false ideas gained by early methods and inadequate facilities, as well as the acquisition of new facts, should continually be kept in mind. The otological clinic in Washington University, for the purpose of development along these lines, has secured and is using for auditory acuity tests in the clinic the various types of audiometers which are available. The standard voice and tuning fork tests are also used, comparisons made and variations noted. This study is a report of this more or less general type of work.

DISCUSSION.

DR. GORDON BERRY: The manufacture and sale of audiometers by large concerns does not always adapt itself to specific needs. A laboratory group is more able to adapt its instruments to required changes. The Bell Telephone Laboratories have done the profession an immense service in carrying out testing on a large scale. Different types of audiometers have their limitations; thus the 3-A audiometer is a simple testing device, but would probably serve better if it offered more amplification with a mean low, high and middle tone, instead of the universal tone it furnishes. The 4-A audiometer is admirable for testing numbers of school children, and useful in doctors' offices and in

clinics. The 2-A audiometer is made for the otologist. It is quicker and simpler than the 1-A but it is apt to get out of order and takes time and money to repair. If we could attain a wider usage, a cheaper production and a more efficient servicing, more men would use audiometers and realize their indispensable value to the profession.

DR. DOUGLAS MACFARLANE: Audiometers are a valuable addition to our armamentarium, and I feel we cannot do without them. Dr. Bunch has shown they are practical instruments for routine use, not merely instruments for scientific investigation, but the instruments themselves do not supply a knowledge of how to use them and to interpret them.

Faults in technique should not be blamed on the audiometer. When we are dealing with the deafened class we find such great differences between single tone testings and speech hearing testings, that it is impossible to explain them. One cannot tell from a 2A audiometer record what speech hearing may be. The hearing of single tones is a relatively simple mental process as compared with speech hearing and interpretation; the latter is related to the central perceptive sphere. It is also highly important to note the type of mentality of the patient being tested. Is he alert and intelligent, or slow, uncertain, cautious, or illiterate?

Another practical point is the checking up on gradual changes in progressive deafness. While it may not be possible to improve hearing conditions, much can be done from the point of view of psychologic and economic readjustment. If deafness is progressing to a point where there will be incapacity for occupation, the change must be anticipated.

DR. EDMUND P. FOWLER: The results of Dr. Bunch agree with those of us who have done audiometric work. The time has gone when it could be supposed we could make a diagnosis on one set of tests alone. There is no one instrument that can make a complete diagnosis.

DR. C. C. BUNCH (closing): In regard to the relation of these tests to hearing aids, it seems to me that in the type of deafness where the patient hears low tones fairly well and high tones very poorly, it is necessary to have some type of device which will amplify high tones more than low, or a filter in an amplifying circuit which will cut down the low tones, which need less amplification. This can be done in the laboratory, but such devices should be available to the otologist. There is no doubt that the psychic element has much to do with benefits received. With the proper attitude of mind the patient will listen more attentively. Women will often refuse to wear a hearing device because they feel that it makes their deafness more conspicuous.

The Presence of Phagocytic Cells (Histiocytes) in Aural Mucosa. Dr. Ralph A. Fenton, with the Collaboration of Olof Larsell, Ph.D.

Aural histopathology in the past was concerned largely with epithelial and bony changes. The role of connective tissue in immunity was neglected. Difficulties of securing and staining material were present.

So-called "reticuloendothelial" connective tissue cells, ordinarily quiescent, are fixed elements capable of phagocytosis when needed. They are demonstrable in tissues through ingestion of vital stains. These cells, "histiocytes," were not mentioned in otologic histopathology until very recently.

Submucosal connective tissue in childhood is thick, becoming granulation tissue when irritated and overgrown by ciliated epithelium from tube mouth (Wittmaack). Meyer disagrees that tubal connection is necessary, mentioning ciliated change at a distance. Thin connective tissue of adult becomes fibrotic with irritation. Eustachian tube is ciliated; there is much adenoid tissue near pharynx.

Demetriades (1927) stained connective tissue intra vitem with pyrrhol blue. Russi (1930) demonstrated histiocytic distribution in guinea pigs, using trypan blue. Schwarz (1931) found constitutional peculiarities influence amount and distribution of connective tissue in fetal and infant temporal bones. Using Maximow's stains on 202 bones, he demonstrates 10 per cent of connective tissue cells to be histiocytes before fifth fetal month, increasing rapidly in proportion after appearance of foreign matter in tympanic cavity at that time. Histiocytes are very numerous at term.

Meyer (1931) considers "myxomatous" or "fetal" tissue only edematous connective tissue similar to that of nasal sinuses and polypi. Our cat material, vital stained with trypan blue, shows few but constant histiocytes along the Eustachian tube, still fewer near tympanic capillaries. Normal adult human material shows fairly numerous histiocytes in the fibrous submucosal tissue of the tube, fewer in the tympanic submucosa. A vascular aural polyp due to long standing chronic suppuration showed numerous histiocytes. Fibrous changes are more marked in granulations adjacent to cholesteatoma, but active histiocytes are also observed. Role of plasma cells is not yet determined.

Histiocytes first act as phagocytes, then form fibrous tissue. The influence on ear pathology is obvious, and suggests extensive experimental work as well as examination of carefully fixed human material prepared with stains which will demonstrate these structures.

DISCUSSION.

DR. L. W. DEAN: I would like to ask one question. How is it that Dr. Fenton is so certain that the stain he is using proves that the cells are histiocytes?

DR. J. GORDON WILSON: The investigations now going on regarding the role of the histiocytes unveil a little more the secret of tissue resistance to disease. Dr. Fenton has, on several occasions added to our knowledge. We hope that he will continue this work. The wandering and sessile histiocytes widely distributed about the body are no doubt as active in diseases of our specialty as they are in general maladies. Regarding their role we can ask a number of unanswerable questions, but I am content to wait until Dr. Fenton can present us with more reliable information.

DR. FENTON (closing): If the histiocyte gets too full of bacteria the cell dies. It is taken up by other cells, broken down or carried away. We checked this work on cats in the frontal sinus mucosa. It is probably the same in the Eustachian tube. Dormant cells become active and take up their histiocytic function. In one specimen which was carefully dissected, there was extrusion and diapedesis into the lumen of the cavity. In Hilding's moving pictures of mucosal cells it is shown that debris is absorbed in various ways by the blood stream or by polymorphonuclears.

We should definitely agree on terms to be used in this application of newer staining methods. The motto for the cytologist of the future will be *semper mutabile*.

DR. DEAN: How soon do you kill the animal after using the vital stain?

DR. FENTON: We have used different times, depending on the size of the animal, and the character of the experiment, but generally within from 24 to 58 hours.

Improved Hearing in Fixation Deafness. Case Reports; Comments. Dr. Eugene R. Lewis, Los Angeles.

Several unusual observations new to me are noted in cases of progressive deafness, and suggest that recovery of a useful degree of hearing may be obtained. These cases of bilateral, progressive deafness of fifteen to twenty years' duration and on the increase, have, by systemic measures only, shown striking improvement.

These facts suggest certain clinical and biological considerations, namely: (1) that deafness was produced by tissue changes which altered shape, size or texture of bony or ligamentous structures at or near the oval window; (2) clinical evidence shows gradual increase of such tissue changes; (3) the bilateral nature of this deafness suggests predisposition to such changes; (4) the bilateral nature of this deafness points to activating systemic causes, such as puberty or pregnancy; and (5) attempts to alter constitution of the body tissues in long standing ear conditions have given striking results such as vertigo, nausea, cessation of tinnitus, increased hearing acuity accompanied by general systemic changes in body weight, blood pressure, blood counts and metabolic rates.

The explanation of these phenomena must be sought in biologic changes which occur after tissue and nutritional factors have been altered. There must

be also *intrinsic* and *extrinsic* factors which take part in the tissue changes under consideration.

The nature of some of these biologic factors may be described as follows: Experiments on regeneration of cell structure in plants and animals, by various workers, have shown that a water removable substance takes part in normal cell division; that oxygen must be extracted from the air to facilitate cell division. Also, in growing organisms, action currents flow from older to younger growing portions of an organism. Action currents of abnormal cells (such as tumor cells) differ from those of normal healthy growing cells. In communities of living cells, differences of potential level are manifested as action currents. In polycellular organisms, electropolarity determines their direction of growth. If the polarity is reversed, the direction of growth is reversed. Heat and chemical action are also seen to have their influence on cell mechanism. Replacement of body cells seems to take place on a one-for-one basis, the cell which is being replaced apparently having some control on the new one. This type replacement can be disturbed by upset of the endocrine balance.

A seed is capable of building into organismal pattern the material it derives from the surrounding soil. In some soils it cannot grow, and the plant varies according to the nature of the soil which surrounds it. Certain soils produce marked differences in plants and their chemical constituents. Thus animals fed on the same type of plants, grown in different localities, may also be affected.

In human beings and animals, the tissues show a tendency to return structures to normal, as in replacement of lost bone structure, in humans, and replacement of dentine and enamel in teeth of calcium deficient animals.

Nature has a strong tendency toward restoring a lost balance to normal pattern. This cell chemistry and tissue replacement tend to restore the constitution of vegetable and animal tissue.

The deafness we have had under consideration is due to mechanical impairments of movable parts of the sound conducting mechanism.

Summary: The cases reported show, first, *normal mobility;* second, *increasing immobility;* third, *decreasing immobility* of moving parts of this mechanism.

There are biologic as well as clinical reasons for attributing the loss of mobility to physical changes in tissues in the region of the oval window and the same reason for attributing the later loss of immobility to changes of a similar nature. Biological studies have revealed the mechanism of these tissue changes. The clinical phenomena herein presented are explicable on this basis.

DISCUSSION.

Dr. Edmund P. Fowler: Dr. Lewis is striving to apply scientific facts and fancies to the varying of biochemistry and tissue changes, in the hope that by trial and error, he will succeed in changing local and general conditions. He reports favorably on two cases treated. Before we can pass judgment on the effects of his treatment we must know the exact amount of hearing and the rate, variations and degree of loss over the fifteen or twenty years during which the deafness is reported to have increased; the family and personal history, and general otologic findings. If, after studying these, we are convinced that these cases are stapes ankylosis, and that real improvement has occurred, two main causes for improvement suggest themselves: first, the one suggested by Dr. Lewis, that is, recession of lesions causing obstructive deafness; second, the improvement in general health and cessation of tinnitus. As far as is known, otosclerosis may cease, but we do not know that it has ever recessed or disappeared.

Dr. Arthur W. Proetz: Bilateral affections occurring simultaneously are, as Dr. Lewis suggests, too common to be accidental. While I follow Dr. Lewis' hypothesis that the bilateral incidence of fixation deafness suggests a predisposition to local tissue changes in that systemic factors aggravate or at least permit rigidity in the conduction apparatus, I am not ready to accept the idea that improvement in the hearing is the result of an improvement in the local inflammation. It seems more likely that the tissue here follows a sequence such as occurs in inflammatory reactions, namely, congestion, hypertrophy, hyperplasia and finally atrophy, and that in the atrophic stage there may be less mechanical interference than in the hyperplastic stage, with consequent improvement in hearing.

DR. THOMAS J. HARRIS: This contribution by Dr. Lewis is of great importance. It should receive our support and confirmation. I hope Dr. Lewis will make his studies available to our members, and especially that it will be put in the hands of our Committee on Progressive Deafness. I feel that it deserves place in our program next year for a further consideration of the subject.

DR. RALPH FENTON: The extremely interesting speculations of Dr. Lewis regarding fixation deafness cases which have shown definite improvement after metabolic changes were undertaken suggest that similar experimental treatment should be undertaken by other investigators, to whom, I feel sure, he will be willing to give full details of his methods.

Undoubtedly most of the hearing loss in these cases was due to abnormal disposition of connective tissue, perhaps for reasons inherent to the individual's own cellular setup. Whether such cases come within the classical definition o otosclerosis or not, and whether connective tissue which has become fixed into bone or scar tissue can be altered in reverse, we must applaud the fearless imagination with which Dr. Lewis has sought out methods of applying biochemical stimuli to such individuals. Should it be possible to arrest the resistless progress of faulty calcification by the use of glandular products and appropriate diet, many types of research now going on will have to be scrapped. No doubt the essayist has it in mind to check up accurately on a long series of similar cases, keeping accurate metabolic records along with several years' accumulation of audiograms. Only by such long continued effort will the proof of his rather astonishing conclusions be made finally of record.

DR. EUGENE R. LEWIS (closing): I can assure Dr. Fowler that these cases have been subjected to the most searching scrutiny; they showed the usual signs of fixation of the stapes. I am unable to prove fixation except by functional test methods. Later we may get some autopsy proof. Dr. Fowler referred to its taking years to change a basal metabolic picture. I have seen a basal metabolic rate of —28 change in six or eight months to plus 18.

Dr. Fentol mentioned otosclerosis. That term does not appear in my paper. In my opinion the second case was definitely otosclerotic; both cases showed deafness of the fixation type; that avoids discussing the exact nature of the cause. In the course of their treatments, changes were noted in many general findings: blood pictures, body weights, etc. The second case showed no change in hearing or tinnitus for eight months. She then began experiencing sudden cessation of tinnitus, during which "it sounded as if a window had been suddenly opened." Other cases have reported similar sensations. Dr. Haris mentioned the putting of this method into the hands of the Committee on Progressive Deafness. This paper is an attempt to do just that. The approach to the problem seems of prime importance, whether in a "localistic" or a "generalistic" frame of mind. A patient presents the effect of all factors whether hereditary or acquired, self-contained or environmental, the exigencies of living and all others. The status praesens of the patient represents the product of all these factors. Inquiries must be made into the family and personal histories, conditions past and present, nutritional, exercise, domestic psychic, emotional, business, environmental—anything tangible, that can be pressed into service.